THE American JOURNAL OF Surgery

SIXTY-SEVENTH YEAR OF PUBLICATION

A PUBLICATION OF THE *Yorke* GROUP

JULY 1958

JULY 1958

THE American JOURNAL OF Surgery

Editor ROBERT M. ZOLLINGER, *Columbus, Ohio*

PROFESSOR AND CHAIRMAN, DEPARTMENT OF SURGERY, OHIO STATE UNIVERSITY, COLUMBUS

Editor Emeritus THURSTON SCOTT WELTON, *Brooklyn, N. Y.*

CLINICAL PROFESSOR EMERITUS OF OBSTETRICS AND GYNECOLOGY

STATE UNIVERSITY OF NEW YORK COLLEGE OF MEDICINE, NEW YORK

ADVISORY BOARD

HENRY T. BAHNSON, *Baltimore*
JOHN M. BEAL, *New York*
CLAUDE S. BECK, *Cleveland*
H. GLENN BELL, *San Francisco*
CLARENCE J. BERNE, *Los Angeles*
B. MARDEN BLACK, *Rochester*
DWIGHT E. CLARK, *Chicago*
RANDOLPH LEE CLARK, JR., *Houston*
H. WILLIAM CLATWORTHY, JR., *Columbus*
FREDERICK A. COLLER, *Ann Arbor*
OSCAR CREECH, JR., *New Orleans*
J. ENGLEBERT DUNPHY, *Boston*
DANIEL W. ELLIOTT, *Columbus*
EDWIN H. ELLISON, *Columbus*
EGBERT H. FELL, *Chicago*
WILLIAM T. FITTS, JR., *Philadelphia*
JAMES D. HARDY, *Jackson*
J. HARTWELL HARRISON, *Boston*
STANLEY O. HOERR, *Cleveland*
EMILE F. HOLMAN, *San Francisco*
CHARLES A. HUFNAGEL, *Washington*
WALTER G. MADDOCK, *Chicago*
K. ALVIN MERENDINO, *Seattle*
CARL A. MOYER, *St. Louis*
WILLIAM H. MULLER, JR., *Charlottesville*
D. W. GORDON MURRAY, *Toronto*
MERLE M. MUSSELMAN, *Omaha*
H. C. NAFFZIGER, *San Francisco*
SIDNEY W. NELSON, *Columbus*
VICTOR RICHARDS, *San Francisco*
JOHN A. SCHILLING, *Oklahoma City*
JOHN C. ULLERY, *Columbus*
ROGER D. WILLIAMS, *Columbus*
HAROLD A. ZINTEL, *New York*

The American Journal of Surgery is published monthly by The American Journal of Surgery, Inc., 11 East 36th Street, New York 16, N. Y. Yearly Subscription, $15.00 U. S. A.; $16.00 Canada; $17.00 Foreign including Pan-American Countries. Single Numbers $2.00; Special and Symposium Numbers $4.00. Second-class mail privileges authorized at New York, N. Y. with additional second-class mail privileges authorized at York, Pa.—New Series, Volume 96, No. 1.
Copyright ©, 1958, by The American Journal of Surgery, Inc.

MANUSCRIPTS: All manuscripts should be typewritten double space and addressed to the Editorial Office of the Journal, 11 East 36th Street, New York 16, N. Y. The top should be indicated on the back of each photograph. *Style for bibliography:* DOE, J. J. Modern technics in surgery. Am. J. Surg., 5:93, 1950.

Change of address must reach us one month preceding month of issue.

ADVERTISING REPRESENTATIVES

New York: P. A. Porter, P. D. Brewer, H. S. Schultz—MUrray Hill 3-2980

Chicago: R. H. Andrew, C. P. Haffner—FRanklin 2-3861

AN AMES CLINIQUICK™

CLINICAL BRIEFS FOR MODERN PRACTICE

what diagnosis should be considered in a male over 50 with unilateral hip pain or low back pain as a dominant symptom?

Prostatic carcinoma

Source—Malament, M.: J. M. Soc. New Jersey *54*:115, 1957.

what per cent of prostatic carcinomas are estimated to be operable at the time of first examination?

Only 5 per cent

Source—Harrison, J. H.; Leman, C., and Herman, H.: Postgrad. Med. *20*:457, 1956.

palliative of choice in prostatic carcinoma

STILPHOSTROL® TABLETS • AMPULS

Diethylstilbestrol Diphosphate, AMES

"...benefits an appreciable percentage of patients with advanced prostatic carcinoma, even after other estrogens have failed."*

Relieves pain, diminishes urinary symptoms, promotes well-being.

*Flocks, R. H.; Marberger, H.; Begley, B. J., and Prendergast, L. J.: J. Urol. *74*:549, 1955.

For complete information write to: Medical Department

AMES COMPANY, INC • ELKHART, INDIANA • Ames Company of Canada, Ltd., Toronto

44158

shock?

OPERATION IS POSSIBLE WITH

life-saving

Levophed®

bitartrate

natural
antishock
pressor
hormone

Levophed raises blood pressure in seconds, makes the "inoperable" patient in shock operable.

Life-saving Levophed is effective even when transfusions fail. Its dependable vasopressor action converts the "poor risk" patient to a good risk.

"...the most satisfactory form of treatment to date"[1]

1. Briller, S. A.: M. Clin. North America 41:619, May, 1957.

Winthrop LABORATORIES, New York 18, N. Y.

Levophed (brand of levarterenol), trademark reg. U. S. Pat. Off.

The American Journal of Surgery

VOLUME NINETY-SIX NUMBER ONE

Contents • July 1958

EDITORIAL

The Challenge of Emergency Clinics 1

ROGER D. WILLIAMS

SCIENTIFIC PAPERS

Intrarenal Arteriovenous Fistula with Hypertensive Cardiovascular Disease. Review of the Literature 3

FRANK MILLOY, JR., EGBERT H. FELL, ROBERT F. DILLON AND AGENOR M. ZAYAS

A report of arteriovenous fistula of renal vessels of traumatic origin causing hypertension which was relieved by nephrectomy. A review and discussion of the literature is given along with a theoretical consideration of the hemodynamics involved.

Studies on Intra-arterial Heparinization 12

PAUL F. GRYSKA, JACK P. ABBOTT AND MICHAEL E. DEBAKEY

The authors conclude that heparin given intra-arterially for the purpose of regional heparinization must be considered as a systemic anticoagulant. The dangers inherent in its use segmentally in association with arterial surgery must be considered.

Ruptured Aneurysm of the Splenic Artery 18

CHARLES L. JAMES

The tenth case of ruptured splenic artery aneurysm with survival is reported; and the incidence, pathology, diagnosis and management of these aneurysms are discussed.

The Status of Head and Neck Surgery in Hospitals Approved for Four-Year Residency Training 24

ARTHUR G. JAMES

This article is concerned with the results of a survey which was prepared in an effort to evaluate the teaching and training in head and neck surgery. The types of procedures which were carried out on general surgery, plastic surgery, and ear, nose and throat surgery are compared.

Contents Continued on Page 5

TRAVENOL LABORATORIES

announces the Coil Kidney Film...

"MACHINE MIMICS MAN"

This dramatic sound and color film demonstrates, by means of animation and patients, how the Travenol Coil Kidney makes hemodialysis practical in almost every hospital. It shows . . . step-by-step . . . the ease of setting up the Travenol Coil Kidney, and how quickly hemodialysis is available to the patient.

"Machine Mimics Man" also reviews normal kidney function, the basic principles of hemodialysis, and its role in renal insufficiencies and certain systemic poisonings.

For information on scheduling the film, "Machine Mimics Man," write to Film Library, Travenol Laboratories, Inc. Morton Grove, Illinois.

Travenol Laboratories, Inc. Morton Grove, Illinois

A DIVISION OF BAXTER LABORATORIES, INC.

Contents • July 1958

VOLUME NINETY-SIX NUMBER ONE

Management of Goiter in Children 27

CARL P. SCHLICKE AND ROBERT BERGHAN

Experiences in the treatment of twenty-three children under fifteen years of age hospitalized for the treatment of goiter are presented. The occurrence of papillary carcinoma of the thyroid gland in four children is emphasized.

The Chvostek Sign. A Clinical Study. 33

ELMER HOFFMAN

A study of 736 patients demonstrated a positive Chvostek sign in 24.7 per cent of those examined. The highest incidence occurred under the age of thirty years in both sexes. Knowledge of the preoperative presence of the Chvostek sign in thyroid patients in the younger age groups is emphasized.

Restoration of the Thoracic Esophagus with Aortic Homograft Following Palliative Subtotal Esophagectomy 38

FRANK A. ROGERS

An arterial homograft was used as a replacement for a portion of the thoracic esophagus following palliative resection for extensive carcinoma. The immediate functional result was satisfactory.

Ruptured Pericostal Steel Wire Retention Sutures. A Post-thoracotomy Complication 43

WALTER H. MASSION, MICHAEL T. LATEGOLA AND JOHN A. SCHILLING

An experimental study is presented demonstrating the hazards of closure of the thoracic wounds with stainless steel retention sutures.

Carcinoma of the Colon and Rectum in Persons under Twenty Years of Age . . 47

M. TISCHER HOERNER

A review of the literature shows that 189 cases of carcinoma of the rectum and seventy-three instances of malignant disease of the colon in patients under twenty years of age have been recorded. The clinical course of this lesion in young people is discussed.

Adenocarcinoma of the Small Intestine 54

ALLAN E. GILBERT AND ROBERT A. WISE

The clinical syndrome presented by carcinoma of the small intestine is illustrated by three reported cases. The diagnostic significance of persistent occult blood in the stool and anemia with no demonstrable disease in the stomach and colon is emphasized, and the principles of surgical treatment are reviewed.

Contents Continued on Page 7

NOTABLY SAFE AND EFFECTIVE INHALATION ANALGESIA

"Trilene"®

Brand of trichloroethylene U.S.P. (Blue)

SELF-ADMINISTERED WITH THE

"Duke"® University Inhaler

No. 3160 Model-M

With the "Duke" University Inhaler, "Trilene" analgesia can be self-administered by the patient, adult or child, under medical supervision, with a relatively wide margin of safety. Induction of analgesia is usually smooth and rapid. Inhalation is automatically interrupted if unconsciousness occurs. Outpatients can generally leave the doctor's office or hospital within 15 to 20 minutes.

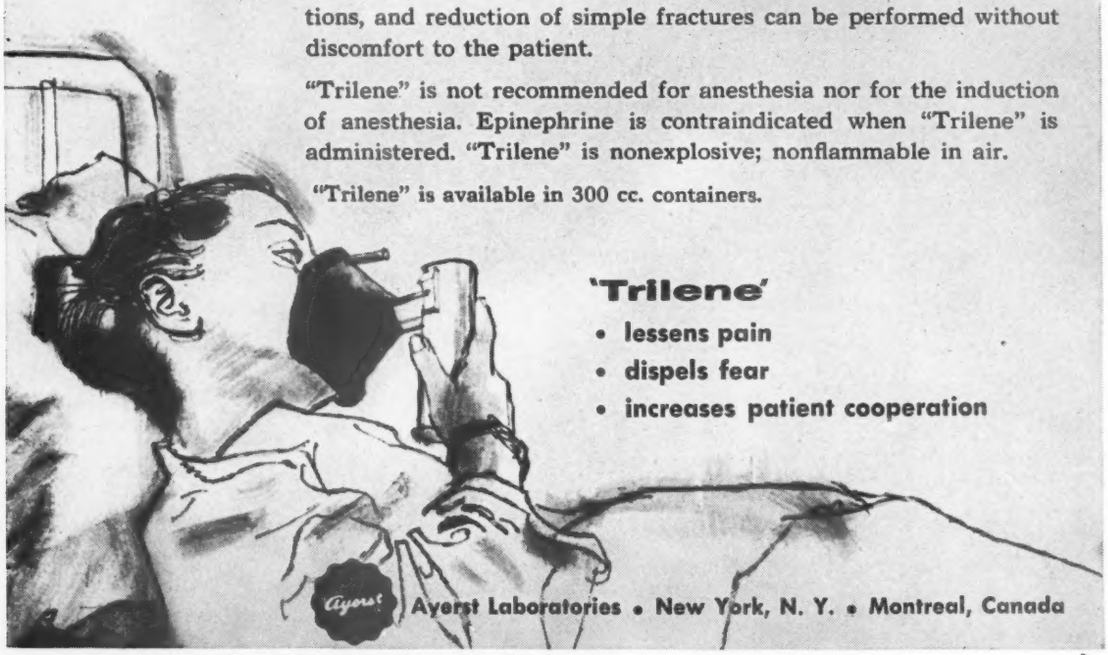
In Obstetrics . . . Self-administered "Trilene" will usually give effective relief of pain throughout labor and, for delivery, it may be employed in association with pudendal block or low spinal anesthesia.

In Pediatrics . . . The "Duke" University Inhaler is so easy to operate that even a child can administer his own "Trilene" analgesia.

In Surgery . . . With "Trilene," self-administered, many so-called "minor" yet painful procedures such as biopsies, suturing of lacerations, and reduction of simple fractures can be performed without discomfort to the patient.

"Trilene" is not recommended for anesthesia nor for the induction of anesthesia. Epinephrine is contraindicated when "Trilene" is administered. "Trilene" is nonexplosive; nonflammable in air.

"Trilene" is available in 300 cc. containers.



'Trilene'

- lessens pain
- dispels fear
- increases patient cooperation

Ayerst Laboratories • New York, N. Y. • Montreal, Canada

Ayerst Laboratories make "Trilene" available in the United States by arrangement with Imperial Chemical (Pharmaceuticals) Limited.

5717

Contents • July 1958

VOLUME NINETY-SIX NUMBER ONE

Atypical Symptom-Sign Complex of Acute Pancreatitis 61

J. L. DONHAUSER AND NOLTON H. BIGELOW

Pain may be entirely absent or insignificant despite the presence of severe pancreatitis. The value of amylase determinations in any type of upper abdominal disorder of uncertain or obscure nature is emphasized to enhance the accuracy of diagnosis.

Acute Emphysematous Cholecystitis. A Case Report and Review of the World Literature. 66

ALFRED EDINBURGH AND ABRAHAM GEFFEN

The authors document the fiftieth case reported of acute emphysematous cholecystitis. They also review cases previously reported, review and discuss the pathogenesis and clinical course of this condition, and evaluate the methods of treatment.

Vaginal Plastic Surgery Based on a New Concept of the Anatomy of the Female Genital Tract. VI. Surgical Treatment of the Lacerated Perineum of Long-Standing. 76

HERMAN SHANN

The author discusses the surgical principles and operative technic of perineoplasty in the treatment of the lacerated perineum of long-standing.

Key Graft Fixation in Mid-tarsal Fracture Dislocation 81

ELIHU FRIEDMANN

A new technic for mid-tarsal arthrodesis by means of a tibial key graft is presented as a method of dealing with fracture dislocations in this area. An illustrative case is reported.

Current Therapy of Elephantiasis of the Lower Extremity 84

THOMAS J. BAKER, JR.

We are given a short review of the surgical treatment of elephantiasis, with the author's operative technic. Present concepts are offered, with stress placed on the patient's willingness to adapt to a "new way of life."

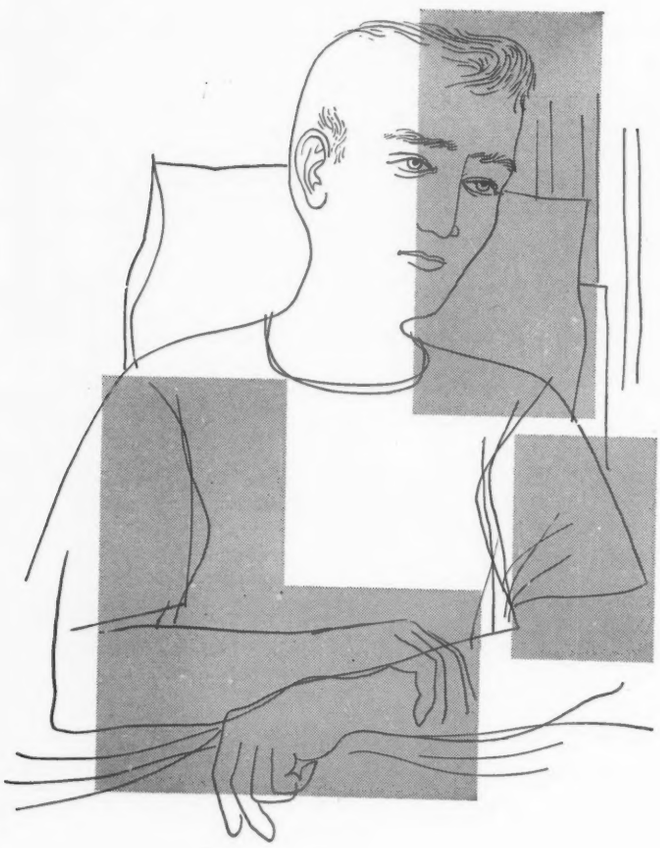
Simplified Herniorrhaphy in Infants and Young Children 88

WILLIAM HOFFMAN

The author suggests that in performing herniorrhaphy in infants the cord and sac be reached directly through an opening made along the fibers of the external oblique fascia without opening the external ring.

Contents Continued on Page 9

Before and after surgery



effective for anxiety, tension and insomnia

- reduces preoperative fear
- helps ensure natural sleep without morning hang-over
- no significant lowering of blood pressure
- no respiratory depression
- well tolerated, relatively nontoxic

Miltown®

THE ORIGINAL MEPROBAMATE
DISCOVERED & INTRODUCED BY
WALLACE LABORATORIES
NEW BRUNSWICK, NEW JERSEY

REFERENCES:

1. *Rushia, E. L.: Preliminary report on the use of meprobamate for pre-anesthetic sedation. J.M.A. Georgia* **46**:*93, March 1957.*
2. *Lamphier, T. A.: The role of meprobamate in postoperative surgical care. Ann. New York Acad. Sc.* **67**:*810, May 9, 1957.*
3. *Kern, E. C.: Preoperative sedation with meprobamate (Miltown) in surgery of eye, ear, nose and throat. Eye, Ear, Nose & Throat Month.* **36**: *408, July 1957.*

CM-6086

Contents • July 1958

VOLUME NINETY-SIX NUMBER ONE

PRACTICAL SURGICAL SUGGESTIONS

An Appraisal of Temporary Gastrostomies and Enterostomies 90
CHARLES B. RIPSTEIN AND KEITH M. SCHNEIDER

A Simplified Technic of Operative Cholangiography 94
GILBERT L. WRIGHT

The Intraperitoneal Landmarks in McBurney Appendectomy 96
JAMES R. WATSON

CASE REPORTS

Gastrojejunocolic Fistula with Associated Islet Cell Tumors of the Pancreas . . 98
KENNETH H. MACGREGOR

A case report of a gastrojejunocolic fistula secondary to peptic ulcer associated with multiple islet cell tumors of the pancreas is presented. Associated hyperinsulinism would indicate mixed islet tumors, i.e., insulomas and ulcerogenic islet cell tumors.

Acute Myasthenia Gravis Following Surgery for Acute Dissecting Aortic Aneurysm under Nitrous Oxide, Pentothal-Curare Anesthesia 102
W. DEAN WARREN, DOUGLAS W. EASTWOOD AND WILLIAM H. MULLER, JR.

A satisfactory repair of an acute dissecting aneurysm of the aorta utilizing the technic of DeBakey is described. The patient died on the fourteenth postoperative day from acute myasthenia gravis. The sensitivity of a patient with myasthenia gravis to anesthesia, especially curare, is discussed.

Paraesophageal Bronchiogenic Cyst 107
ERNEST E. SCHNOOR AND JOHN E. CONNOLLY

This is the fourth verified case of paraesophageal bronchiogenic cyst. Theories of development, pathology and surgical consideration are discussed.

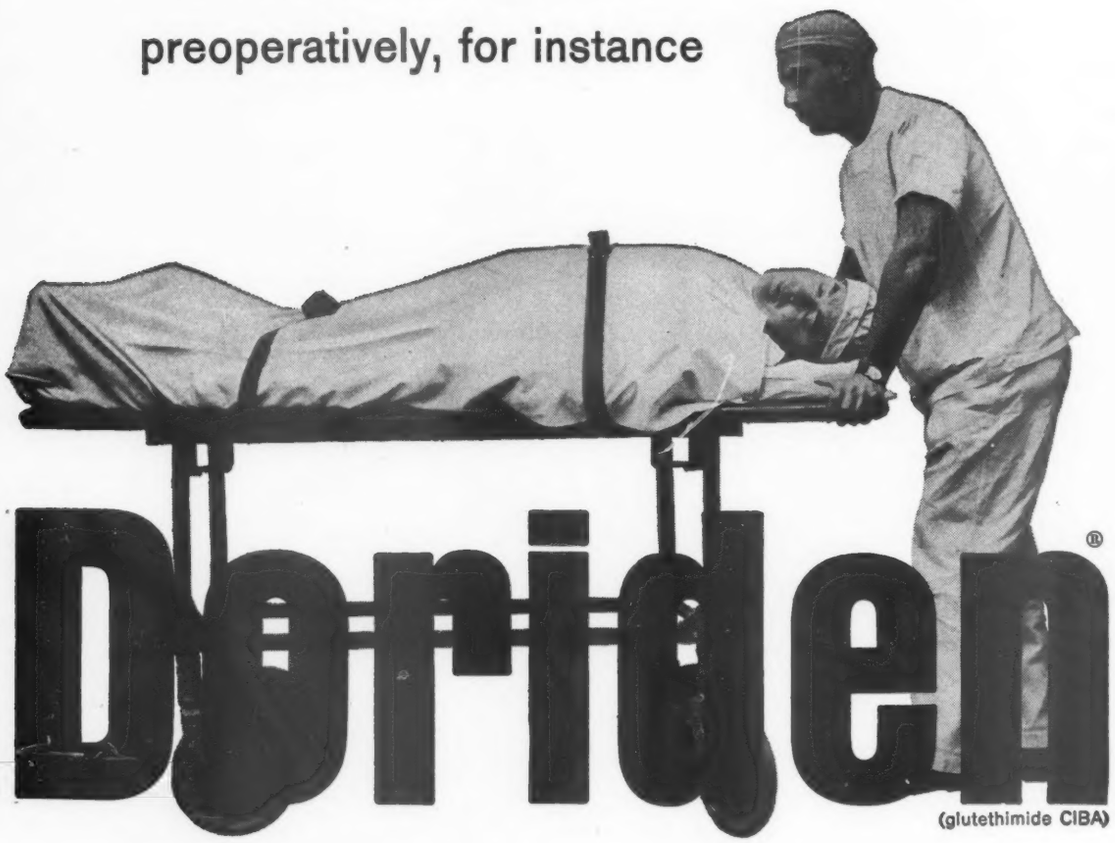
Hemorrhage from Esophageal Varices—Its Prognostic Significance 110
EMILE L. MEINE, JR. AND LEON J. WITKOWSKI

The authors give a short review of the subject, enumerate the patients under their care for a period of five years, and then record case reports intended to show the complexity of the problems of acute esophageal bleeding in widely different age groups as well as the difficult and sometimes frustrating surgical problems involved.

Contents Continued on Page 11

Less restricted* sedation . . .

preoperatively, for instance



Doriden®

(glutethimide CIBA)

"One hour prior to surgery, twenty-five patients were given one-half to one gram of Doriden. Ten of the twenty-five patients slept on the stretcher while waiting to be wheeled into the operating room. . . . the remaining patients seemed to have the calm indifference that is found in partially narcotized patients. There was no respiratory depression. The drug provided very satisfactory preoperative sedation."[1]

*SEDATION WITH DORIDEN IS LESS RESTRICTED because, unlike barbiturates, it is not contraindicated where renal and hepatic disorders are present; unlike many barbiturates, Doriden rarely causes pre-excitation; unlike barbiturates traditionally used for sedation, Doriden is metabolized quickly, thus rarely produces "hangover" and "fog."

SUPPLIED: TABLETS, 0.125 Gm., 0.25 Gm. and 0.5 Gm.

1. Matlin, E.: M. Times 84:68 (Jan.) 1956.

CIBA SUMMIT, N. J. 2/2497MK

Contents • July 1958

VOLUME NINETY-SIX NUMBER ONE

Delayed Postgastrectomy Hemorrhage from Ruptured Traumatic Aneurysm . . 115

CLIFTON F. WEST, JR.

A delayed fatal hemorrhage following an antecolic Polya type of resection resulted from a traumatic aneurysm produced by injury to the major gastroduodenal artery. The cause of the hemorrhage was not discovered at the time of the secondary operation since the lesion was obscured by the rolled-over anterior wall of the duodenal stoma.

Surgical Importance of Bile Duct Adenoma of Liver 118

HUGH F. LUDDECKE AND ILL LYONG YOON

Two cases of bile duct adenoma found at autopsy are reported. The gross similarity of this lesion to metastatic carcinoma is stressed. It is urged that a liver nodule discovered at the time of operation should be examined by a frozen section.

Volvulus of the Transverse Colon 122

SCOTT J. BOLEY

The literature on volvulus of the transverse colon has been reviewed. Two new cases of this rare entity are reported. The author emphasizes that etiologic factors are almost always present and must be found in order to insure adequate surgical therapy.

Carcinoma of the Common Bile Duct 126

DONALD C. GEIST

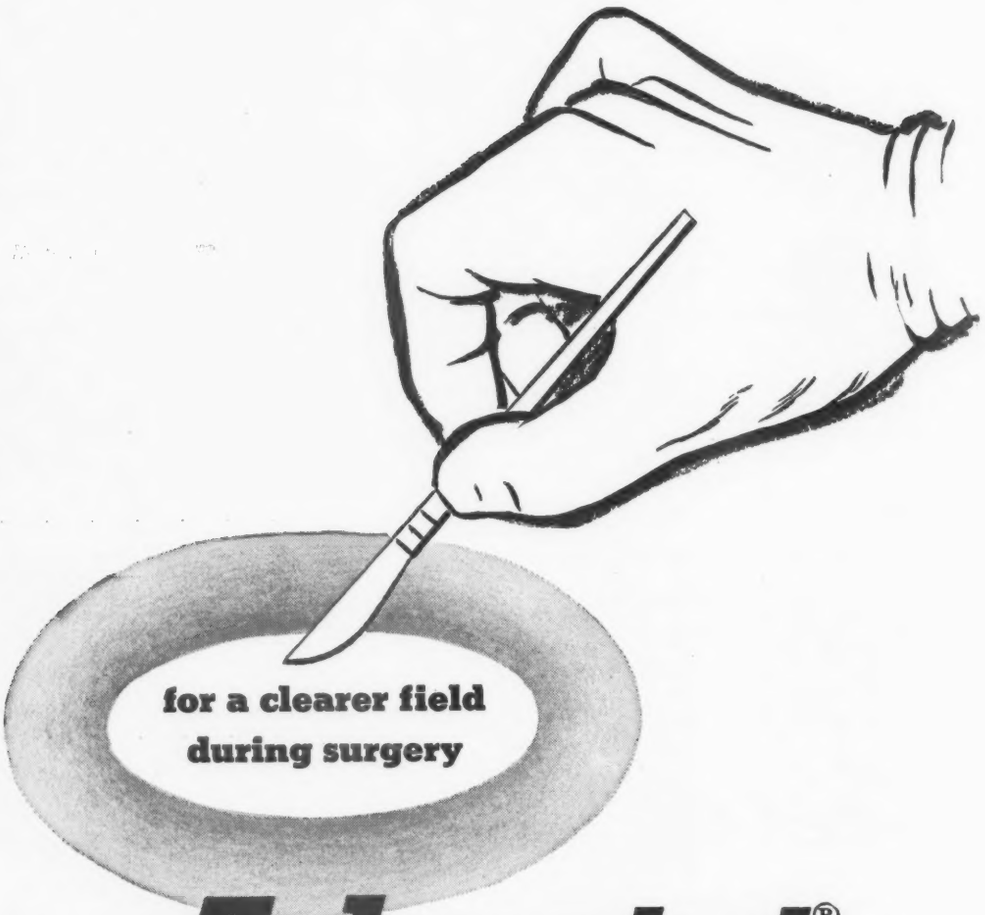
A case is presented of carcinoma of the common bile duct. The patient is alive and well three years following a palliative local resection.

An Unusual Late Sequela Following Gastrectomy 129

THOMAS C. CASE

The report of a case of an unusual type of internal hernia which followed a gastric resection. The author concludes that closure of the opening should be considered as a routine procedure.

BOOK REVIEWS 131



Adrestat® ORGANON

A COMPLETE SYSTEMIC HEMOSTAT

Adrestat complements surgical skill with effective hemostasis. It promotes retraction of severed capillary ends and reduces capillary bleeding and oozing; prevents or corrects abnormal capillary permeability and fragility; prevents delayed clotting time and bleeding due to hypoprothrombinemia. Yet Adrestat has no effect on the normal constituents and process of blood clotting.

AVAILABLE IN THREE DOSAGE FORMS:

ADRESTAT Capsules and Lozenges for pre- and post-operative use, each containing:

Adrenochrome Semicarbazone, 2.5 mg. (present as Carbazochrome Salicylate*, 65.0 mg.); Sodium Menadiol Diphosphate (Vitamin K Analogue), 5.0 mg.; Hesperidin, Purified, 50.0 mg.; Ascorbic Acid, 100.0 mg.

Capsules in bottles of 30; Lozenges in boxes of 20

ADRESTAT (F) 1-cc ampuls for use during surgery, each containing Adrenochrome Semicarbazone (present as Carbazochrome Salicylate*, 130.0 mg.)

Boxes of five 1-cc ampuls

*Pat. Nos. 2,581,850; 2,506,294

Organon inc.

ORANGE, N. J.

all expendable syringes are not the same

there is a difference

STYLEX™

the only PRECISION *expendable syringe*

Only Pharmaseal, with years of experience in research and development of quality plastics for the medical profession, could produce the STYLEX syringe — the ultimate in safety and precision.

Every STYLEX syringe is guaranteed for safety and performance.

ONLY THE FINEST PRODUCTS BEAR THIS NAME

PHARMASEAL®

PHARMASEAL LABORATORIES • *Affiliate of Don Baxter, Inc.* • GLENDALE 1, CALIFORNIA

® Trademark

Metabolic demands increase when the body is subjected to surgical procedures, burns, fractures and illness. Under such circumstances, restitution of depleted vitamin reserves will accelerate body repair. STRESSCAPS provide essential water-soluble vitamins in a professionally accepted formulation.

STRESSCAPS IN STRESS

- **Infection** • **Physiologic Trauma** • **Endocrine Dysfunction**
- **Emotional Stress** • **Pre- and Postoperatively**

Each Capsule Contains:

Thiamine Mononitrate (B_1)	10 mg.
Riboflavin (B_2)	10 mg.
Niacinamide	100 mg.
Ascorbic Acid (C)	300 mg.
Pyridoxine HCl (B_6)	2 mg.
Vitamin B_{12}	4 mcgm.
Folic Acid	1.5 mg.
Calcium Pantothenate	20 mg.
Vitamin K (Menadione)	2 mg.

Average Dose: 1-2 capsules daily.

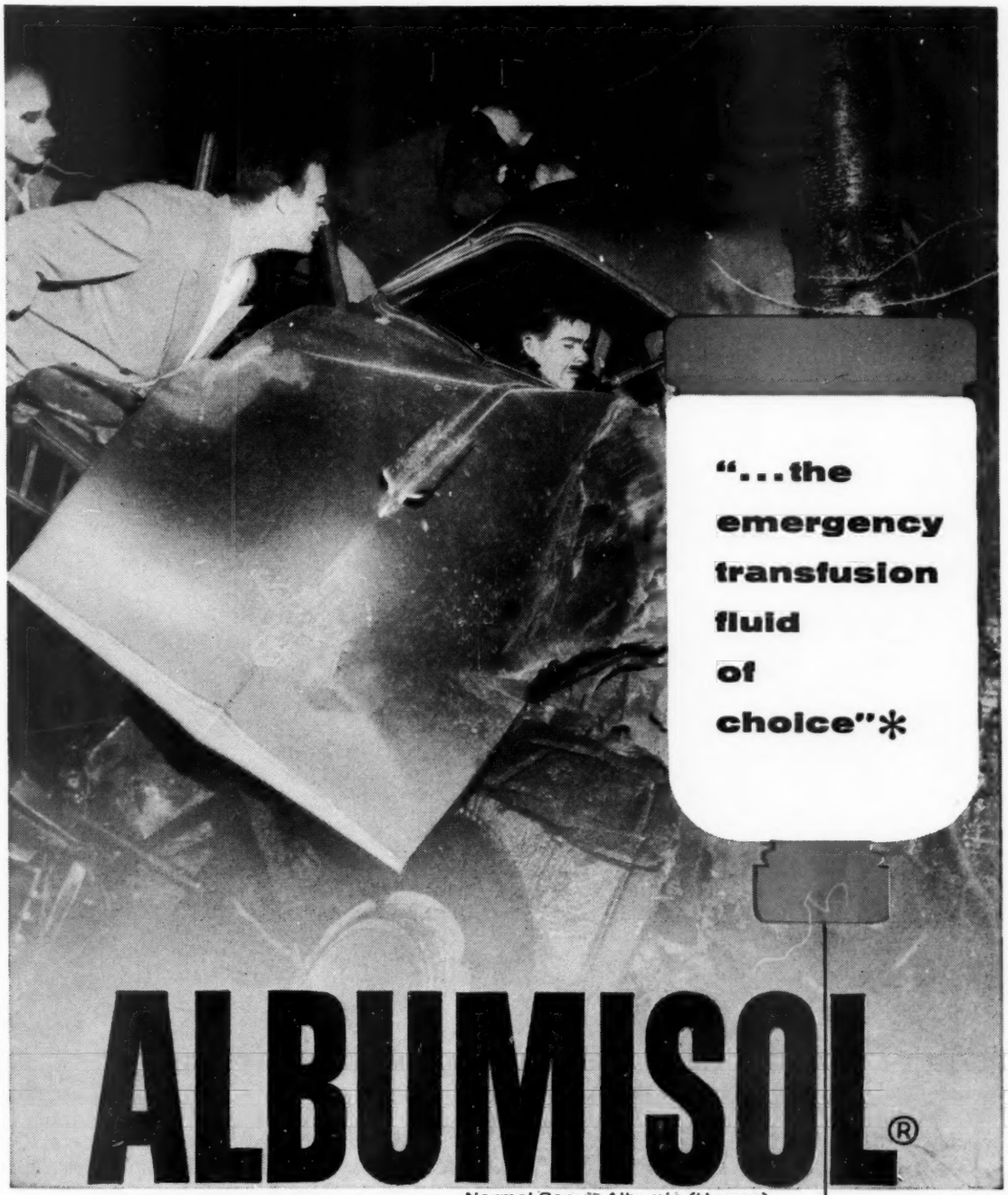
STRESSCAPS*

STRESS FORMULA VITAMINS LEDERLE

Lederle

LEDERLE LABORATORIES DIVISION, AMERICAN CYANAMID COMPANY, PEARL RIVER, NEW YORK

*Reg. U. S Pat. Off.



ALBUMISOL®

Normal Serum Albumin (Human)

No risk of serum hepatitis
Ready for administration; no storage problems
Human protein; readily metabolized
Contains no blood-clotting components
No grouping, typing, cross-matching required

Supplied: 'ALBUMISOL' 5%—in 250 and 500 cc. bottles in packages with a set of disposable intravenous equipment.

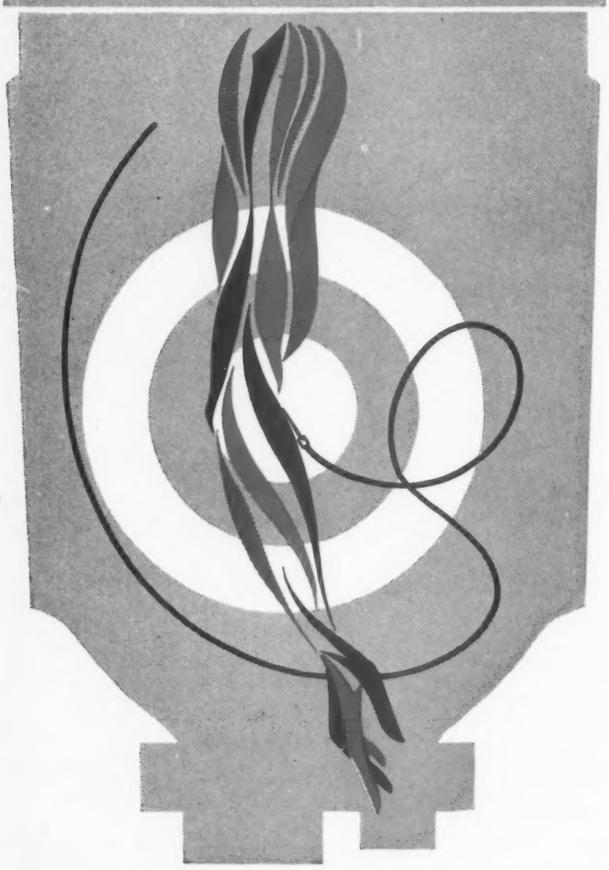
Also supplied: 'ALBUMISOL' 25% (Salt-Poor)—in 20 cc. bottles; in 50 cc. bottles in packages with a set of disposable intravenous equipment.

*Janeway, C.A.: Quart. Rev. Med. 9:153 (Aug.) 1952.

MSD

MERCK SHARP & DOHME
Division of MERCK & CO., INC.
Philadelphia 1, Pa.

HOMEOLYTES / *meet needs precisely*



less want

less waste

less work

HOMEOLYTE®

Multipurpose Essential Electrolyte

SOLUTIONS

Because the Homeolyte dosage system is based on the individual patient's needs and tolerances the Homeolytes provide optimal amounts of water and all essential electrolytes. Thus, further derangement of fluid balance via water or electrolyte excess or inadequacy, with undesired retention or diuresis, does not occur as with dextrose in water and saline solutions. Homeolytes are useful in most fluid imbalances for maintenance or repair in infants or adults. ***Send for reprint: "Multiple Electrolyte Solutions, their use in Parenteral Fluid Therapy," Parenteral Division, Mead Johnson & Company, Evansville 21, Indiana.***

SOLUTIONS	*Standard · Amigen® · Homeolytes® · Levugen®*
EQUIPMENT	*solution sets · blood sets and bottles*
SERVICES	*therapy guides · teaching aids*

Mead Johnson

Symbol of service in medicine

GUDEBROD NON-ABSORBABLE SUTURES

1st

since 1870

with complete packaging

with all non-absorbable sutures

- Surgeons used Gudebrod silk as suturing material back in 1870.
- In 1899 Gudebrod made the first silk sutures especially for surgical use.
- Gudebrod then applied their years of experience to cotton and in 1940 brought out the first cotton sutures.
- For positive identification by color, Gudebrod produced in 1953 the first COLOR-CODED® cotton sutures.
- A year later, in 1954, Gudebrod advanced by making the first COLOR-CODED® silk sutures.
- In 1956 came the Cerethermic* finish . . . a new standard in strength, smoothness and ease of handling.
- And in 1957, the first all-dry Mintraumatic® suture was introduced.

*T.M.

WHETHER IT'S SILK, COTTON OR NYLON, WHATEVER SUTURE FIBER YOU PREFER, IN WHATEVER PACKAGING BEST SUITS YOUR NEEDS, GUDEBROD HAS IT... CHAMPION ... TOP QUALITY.

Gudebrod BROS. SILK CO., INC.

Surgical Division: 225 West 34th St., New York 1, N. Y.

Executive Offices: 12 South 12th St., Phila. 7, Pa.

CHICAGO • BOSTON • LOS ANGELES

clinical report[1]
on the use of

DESITIN®
OINTMENT

in

BURNS

in 246 patients

- **in first degree burns** 98.88% treated with cod liver oil ointment (Desitin) were completely healed in ten days, in contrast to only 85% of the control group.
- **in second degree burns** 98.18% were healed with Desitin completely in fourteen days, only 85.61% of control group.
- **in third degree burns** all patients treated with Desitin Ointment healed in a gratifying manner in 2 to 4 weeks.

Desitin Ointment helps achieve "early, clean and healthy healing" in traumatic and infected wounds, burns, abdominal fistulas, pressure sores and ulcers, pilonidal cysts and anorectal ulcerations[1] with minimal scarring.

1. Grayzel, H. G. and Schapiro, S.: Western J. Surg. 64:558, 1956.

samples *and complete reprint on request.*

DESITIN CHEMICAL COMPANY
812 Branch Avenue, Providence 4, R. I.

Bristol *introduces*

KANTREX

–a dramatically new

"STUBBORN SPECTRUM"

bactericidal

antibiotic

Announced at two-day conference
under auspices of
NEW YORK ACADEMY OF SCIENCES
JULY 10 AND 11, 1958

Chemical, microbiological, pharmacologic and clinical aspects reviewed in 35 papers by eminent investigators. For a complimentary copy of the complete proceedings, write to

Medical Department
BRISTOL LABORATORIES INC.
630 Fifth Avenue
New York, N. Y.

KAN

—bactericidal

against a wide variety

of recalcitrant organisms

—even against

resistant staph!

TREX™

Kanamycin Sulfate

TYPICAL CASE HISTORIES

FROM "TERMINAL" STATE TO COMPLETE RECOVERY

*in septicemia and pneumonia**

A.B., a 1-year-old girl, had received chloramphenicol, erythromycin, penicillin, and bacitracin without effect, for septicemia and pneumonia due to *Staphylococcus aureus.*

KANTREX was started on the 18th day of her illness in a dosage of 15 mg./Kg. a day for the first day, and 10 mg./Kg. for 25 days, in divided doses. Total dosage for the 26 days was 1.68 Gm.

A "dramatic improvement" was noted immediately after KANTREX was started. Although the patient seemed "terminal" before KANTREX was administered, she showed rapid improvement, and X-ray evidence of the disease gradually disappeared. She was discharged "completely well." No toxic reactions were observed.

---

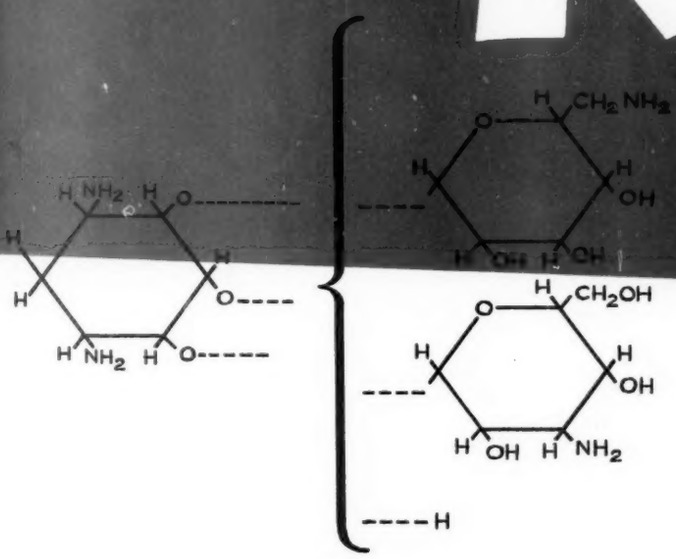
*E. M. Yow and O. Monzon, Dept. of Medicine, Baylor Univ. College of Medicine, Houston, Tex.: Personal communication.

A new "stubl

Clinical studies show that many stubborn organisms resistant to chloramphenicol, penicillin, streptomycin, erythromycin, chlortetracycline, tetracycline, oxytetracycline, oleandomycin, and novobiocin—singly or in combination—are sensitive to KANTREX.

born spectrum" antibiotic–

KAN



KANTREX—an antibiotic derived from *Streptomyces kanamyceticus* —is bactericidal against a wide variety of Gram-positive and Gram-negative organisms, including strains of *Micrococcus pyogenes* var. *aureus*—the most notorious of the "resistant staph."

TREX

Kanamyc

–for intramuscu

TYPICAL CASE HISTORIES

"A THERAPEUTIC TRIUMPH"

*in staphylococcal endocarditis**

L.B., an adult male, was diagnosed as having tricuspid end
multiple pulmonary septic infarcts, due to a penicillin-resi
lococcus aureus hemolyticus. He was acutely ill and, afte
million units of penicillin a day, plus 2 Gm. of erythromy
a "striking downhill course."

Intramuscular kanamycin (KANTREX) was started at a dos
t.i.d. for 3 days, after which it was raised to 1.0 Gm. t.i.d.
for 5 weeks. Following this, the dosage was lowered to 0.5
2 weeks. No evidence of any kind of kanamycin toxicity
during the 7½ weeks of therapy.

A prompt temperature drop occurred by the fourth da
therapy. Subsequently, X-rays showed resolution of the

The patient was discharged as recovered on 2/7/58, and a
up since then showed him to be entirely well. This case wa
the investigator as "a therapeutic triumph."

---

*Sol Katz, District of Columbia General Hospital, Washington, D.C.: Pers

TM

cin Sulfate

ular use

docarditis with
istant *Staphy-*
er receiving 24
ycin, continued

sage of 0.5 Gm.
and continued
5 Gm. t.i.d. for
y was observed

y of KANTREX
septic infarcts.

clinical follow-
as described by

sonal communication.

Among the pathogenic organisms susceptible to KANTREX are various strains of *Staph. aureus* and *albus, Proteus, A. aerogenes, Klebsiella pneumoniae, Salmonella, Shigella, E. coli, paracolobacterium, Pseudomonas* and *enterococcus.*

Preliminary studies indicate that KANTREX is a promising agent against *Mycobacterium tuberculosis,* including streptomycin- and isoniazid-resistant strains.

for resistant organisms—

KANTREX™

Kanamycin Sulfate

Indications

Indications for KANTREX include infections due to kanamycin-susceptible organisms:

Respiratory infections: laryngitis, tracheitis, bronchitis, pneumonitis, broncho-pneumonia, lung abscess, pleuritis, empyema and bronchiectasis.

Urinary tract infections: acute and chronic pyelonephritis, cystitis.

Soft tissue infections: wound infections, abscesses, cellulitis, osteomyelitis, blood stream infections.

Dosage

Adults: Average daily intramuscular dose 1 to 2 Gm. in 2 to 4 equally divided doses.

Children: Average daily intramuscular dose 15 to 30 mg. per Kg. in 2 to 4 equally divided doses.

Precaution

In the course of extensive clinical trials, signs of renal irritation and skin eruptions (which disappeared on cessation of therapy) were occasionally noted. Signs of eighth nerve dysfunction—tinnitus, vertigo and loss of hearing—were observed in a few patients. These patients were predominantly over forty-five years of age; all had received 18 grams or more of KANTREX. In this latter respect it would appear that KANTREX has less toxic potential than streptomycin.

Supply

KANTREX is available in rubber-capped vials as a ready-to-use sterile aqueous solution in two concentrations (stable at room temperature indefinitely):

KANTREX (kanamycin sulfate) 0.5 Gm. in 2 ml. volume.

KANTREX (kanamycin sulfate) 1.0 Gm. in 3 ml. volume.

Also available: KANTREX (kanamycin sulfate) Capsules, 0.5 Gm. for preoperative bowel antisepsis; bottles of 20 and 100.

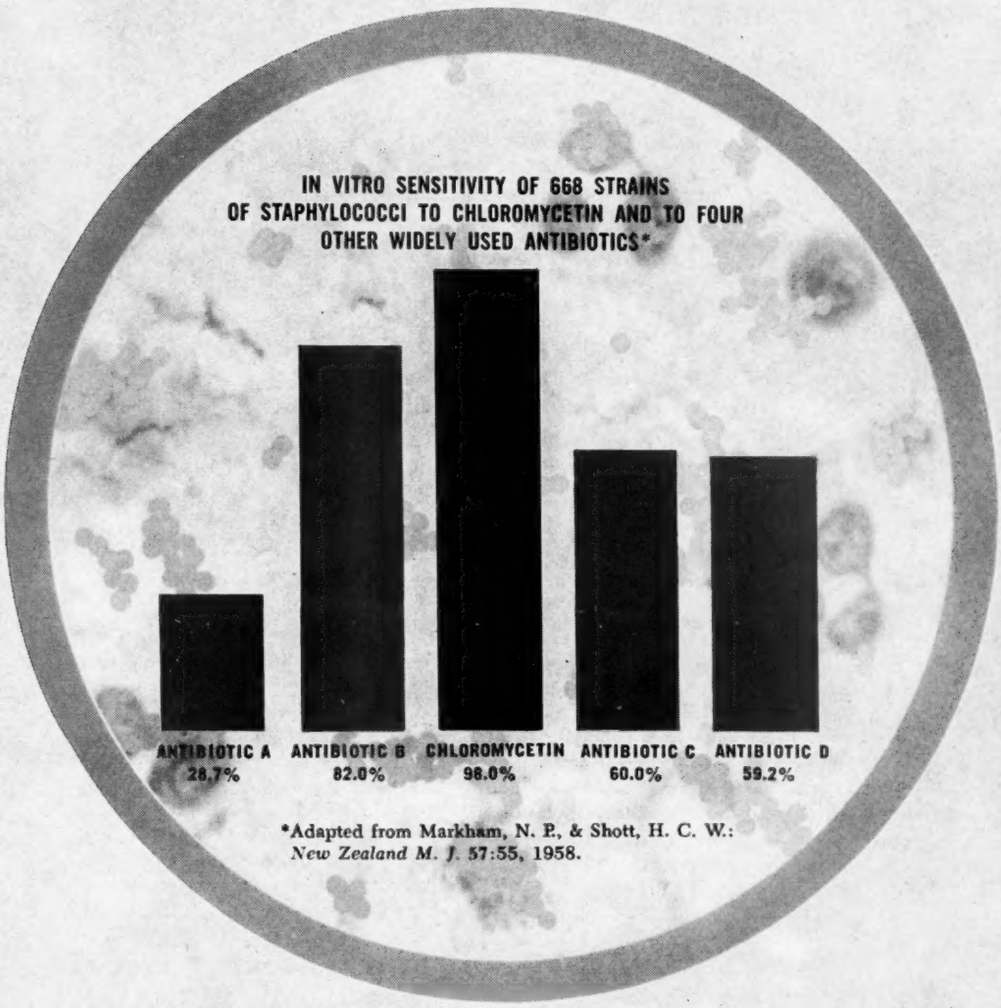
Comprehensive literature available on request

Bristol LABORATORIES INC. • SYRACUSE, NEW YORK

MOST CONSISTENTLY EFFECTIVE AGAINST STAPHYLOCOCCI

CHLOROMYCETIN®

COMBATS MOST CLINICALLY IMPORTANT PATHOGENS


IN VITRO SENSITIVITY OF 668 STRAINS OF STAPHYLOCOCCI TO CHLOROMYCETIN AND TO FOUR OTHER WIDELY USED ANTIBIOTICS*
ANTIBIOTIC A 28.7%
ANTIBIOTIC B 82.0%
CHLOROMYCETIN 98.0%
ANTIBIOTIC C 60.0%
ANTIBIOTIC D 59.2%

*Adapted from Markham, N. P., & Shott, H. C. W.: *New Zealand M. J.* 57:55, 1958.

CHLOROMYCETIN (chloramphenicol, Parke-Davis) is a potent therapeutic agent and, because certain blood dyscrasias have been associated with its administration, it should not be used indiscriminately or for minor infections. Furthermore, as with certain other drugs, adequate blood studies should be made when the patient requires prolonged or intermittent therapy.

PARKE, DAVIS & COMPANY · DETROIT 32, MICHIGAN

50858

MEDICAMENTA VERA · PD & CO.

Reports from Investigators

In medical student volunteers, 'Deaner' produced increased daytime energy and attentiveness at lectures, sounder sleep (with a reduction in the hours of sleep needed), better ability to concentrate on both studying and writing, decreased apprehensiveness prior to and during examinations, a more affable mood and outspoken personality.

1. Murphree, H.B., Jr.; Jenney, E.H., and Pfeiffer, C.C.: 2-Dimethylaminoethanol as a Central Nervous System Stimulant. Presented before Assoc. for Research in Nervous and Mental Disease, New York, Dec. 12-14, 1957. To be published.

In Exhaustion and Depression—In a study of over 100 patients suffering from various psychiatric disorders, especially exhaustion and mild depression, the clinical effect of 'Deaner' was to increase energy and to relieve depression in over 70%.

2. Lemere, F., and Lasater, J.H.: Am. J. Psychiat. *114*:655 (Jan.) 1958.

In Learning Problems—Some of the children with reading problems and other learning defects have improved markedly during their treatment with 'Deaner'

3. Oettinger, L.: To be published.

the Anti Depressant

Deaner™

2-dimethylaminoethanol (deanol)

A Totally New Molecule for the Treatment of

Chronic Fatigue States
Mild Depression
Chronic Headache
Migraine
Neurasthenia

In extensive clinical trials, DEANER has proved to be of value in that large contingent of patients with vague, undefined symptoms, who feel under par and lack energy or are mildly depressed.

Patients with chronic headache including migraine are benefited.

Advantages of 'Deaner'

Effects come on gradually and are prolonged...

Without causing hyperirritability, jitteriness or emotional tension...

Without causing excess motor activity...

Without causing loss of appetite...

Without elevating blood pressure or heart rate...

Without sudden letdown on discontinuance of therapy.

Dosage

Initially, 1 tablet (25 mg.) daily in the morning. Maintenance dose, 1 to 3 tablets (25 mg. to 75 mg.) for adults; ½ to 3 tablets for children. Full benefits may require two weeks or more of therapy.

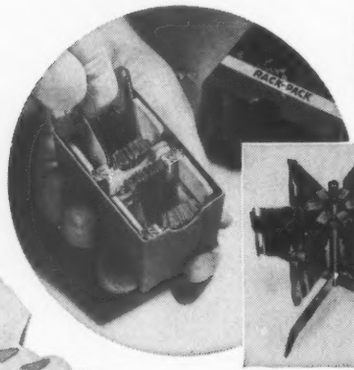
'Deaner' is supplied in 25 mg. scored tablets in bottles of 100.

Another Riker *First*
LOS ANGELES

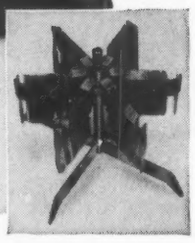
An EDGE on them all

Cutting efficiency and maximum blade performance has always been the surgeon's first consideration when choosing a surgical blade. BARD-PARKER offers you a blade made with the same consideration in mind . . . a blade of *carbon steel* of course . . . so superior for *fine cutting edges.*

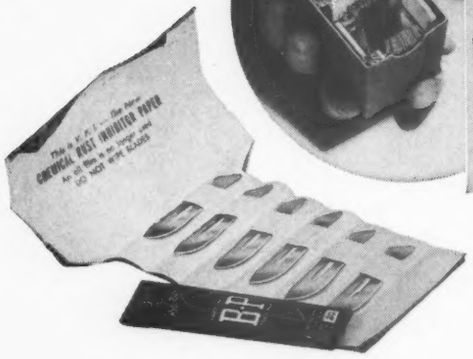
B-P RIB-BACK Blades are now available . . .



in the Puncture Proof Sterile Blade package that can be autoclaved.



in the RACK-PACK package—blades pre-racked ready for sterilization.



in the CONVENTIONAL package—six of one size in a rust-proof wrapper.

It's Sharp

Ask your dealer

B-P **BARD-PARKER COMPANY, INC.**
DANBURY, CONNECTICUT
A DIVISION OF BECTON, DICKINSON AND COMPANY

B-P • RIB-BACK • IT'S SHARP • RACK-PACK are trademarks of BARD-PARKER

WHY RISK DELAYED RECOVERY FROM 'HOSPITAL STAPH' INFECTIONS?

Urinary tract infections, due to staphylococci or proteus (resistant or otherwise), may not respond to any antimicrobial agent except CATHOMYCIN (novobiocin). CATHOMYCIN has a long, established record* of effectiveness against organisms resistant to most other antibiotics. It may be administered in combination with sulfonamides or with other antibiotics, providing a broad spectrum of action and protection against the emergence of resistant strains.

Especially useful for those hard-to-treat urinary tract infections, even those complicated by resistant staphylococci or resistant proteus, CATHOMYCIN is rapidly absorbed—producing therapeutic blood levels with a duration of 12 hours or more. It is generally well tolerated and there is no evidence of cross-resistance with other antibiotics.

CATHOMYCIN®

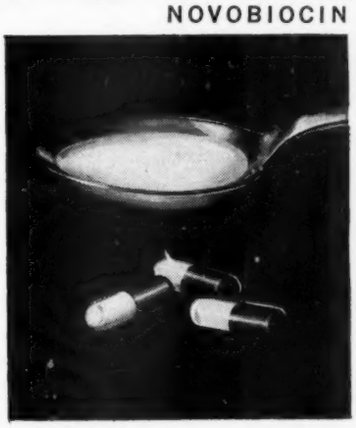
for staphylococcic septicemia, enteritis, postoperative wound infections and other serious staph infections.

NOVOBIOCIN

DOSAGE: Adults: CATHOMYCIN Sodium 2 capsules b.i.d. or CATHOMYCIN Calcium Syrup 4 teaspoonfuls b.i.d. Children: (up to 12 years) 2 to 8 teaspoonfuls daily in divided doses based on 10 mg. CATHOMYCIN per lb. of body weight per day.

SUPPLIED: Capsules sodium novobiocin, each containing the equivalent of 250 mg. of novobiocin—vials of 16 and 100—and as an orange-flavored syrup (aqueous suspension), in bottles of 60 cc. and 473 cc. (1 pint). Each 5 cc. CATHOMYCIN Syrup contains 125 mg. (2.5%) novobiocin, as calcium novobiocin.

*Complete bibliography available on request.



MSD MERCK SHARP & DOHME Division of MERCK & CO., Inc., Philadelphia 1, Pa.

NOTICE TO SURGEONS

The Surgical Products Division, American Cyanamid Company, takes pleasure in announcing a new development of outstanding importance to your hospital...and to your patients.

In the past we have worked with surgeons and hospital personnel to introduce such important product advances as the new all-purpose D & G Elliptron* Surgical Needle, the revolutionary D & G Surgilar® and D & G Surgilope SP* plastic suture packages that give you safer, stronger, more flexible sutures, and a complete line of Vim hypodermic syringes and needles.

Now, in a sweeping move to provide hospitals with faster, more efficient service and reduce hospital costs substantially, Surgical Products Division introduces a plan by which hospitals may purchase all products directly from the manufacturer!

Key personnel responsible for purchasing in your hospital are now being given full details on the DIRECT-PURCHASE PLAN. This exclusive innovation will save the average hospital thousands of dollars a year on quantity purchases...and at the same time give them the added benefit of more efficient service and new rapid delivery provided by our large staff of highly trained representatives and our unparalleled network of branch offices and regional supply centers.

The DIRECT-PURCHASE PLAN is bound to become an important topic of discussion among your hospital associates. We think you'll agree that it marks one more important step in our comprehensive program to bring your hospital the finest products at the lowest possible cost.

NEW MONEY-SAVING DIRECT PURCHASE PLAN FOR HOSPITALS

CYANAMID
AMERICAN CYANAMID COMPANY
SURGICAL PRODUCTS DIVISION
NEW YORK, N. Y.

SALES OFFICE: DANBURY, CONN.

PRODUCERS OF DAVIS & GECK BRAND SUTURES AND VIM® BRAND HYPODERMIC SYRINGES AND NEEDLES.
DISTRIBUTED IN CANADA BY: CYANAMID OF CANADA LIMITED. MONTREAL 16, P.Q.

*Trademark

More
than
enough
Gantrisin
Tablets
to
encircle
the
earth—

More than enough Gantrisin Tablets to encircle the earth-

If all the Gantrisin tablets* produced and used since the introduction of this single, soluble sulfonamide were placed "end to end," the distance would exceed 24,000 miles--more than enough to encircle the globe at the equator.

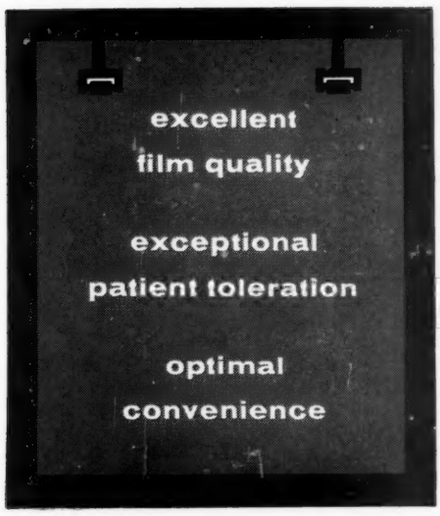
This acceptance by the medical profession is overwhelming evidence of the clinical usefulness, efficacy and safety of Gantrisin.

*More than 3 billion tablets (liquids and other forms not included).

GANTRISIN®—brand of sulfisoxazole (3,4-dimethyl-5-sulfanilamido-isoxazole)

ROCHE LABORATORIES
Division of Hoffmann-La Roche Inc
Nutley 10, N.J.

Original Research in Medicine and Chemistry



RENOGRAFIN

Squibb Sodium and Methylglucamine Diatrizoates

the intravenous contrast medium suited for virtually every urographic and angiographic use

for use in cerebral angiography . . . peripheral arteriography . . . venography: **RENOGRAFIN 60%**

for use in major vasography . . . angiocardiography (pediatric) . . . aortography: **RENOGRAFIN 76%**

for visualization of the kidney and urinary passages, including the renal pelves, ureters, and bladder:
RENOGRAFIN 60% or RENOGRAFIN 76%

for routine retrograde pyelography: **RENOGRAFIN 30%**

Supply: Renografin 76%: 20 cc. ampuls with 1 cc. ampul for sensitivity testing. Renografin 60%: 25 cc. ampuls with 1 cc. ampul for sensitivity testing. Also *Hospital Pak* containing 100 ampuls of 25 cc. (without sensitivity-testing ampul). Renografin 30%: 50 cc. vials.

and now RETROGRAFIN

Squibb Sodium and Methylglucamine Diatrizoates and Neomycin Sulfate

. . . for retrograde pyelography when urinary tract infection is present or suspected

Retrografin provides the same advantages in retrograde pyelography as provided by Renografin 30%, but it also contains 2½% neomycin (as the sulfate) for widely effective bactericidal action in cases with real or suspected infection.

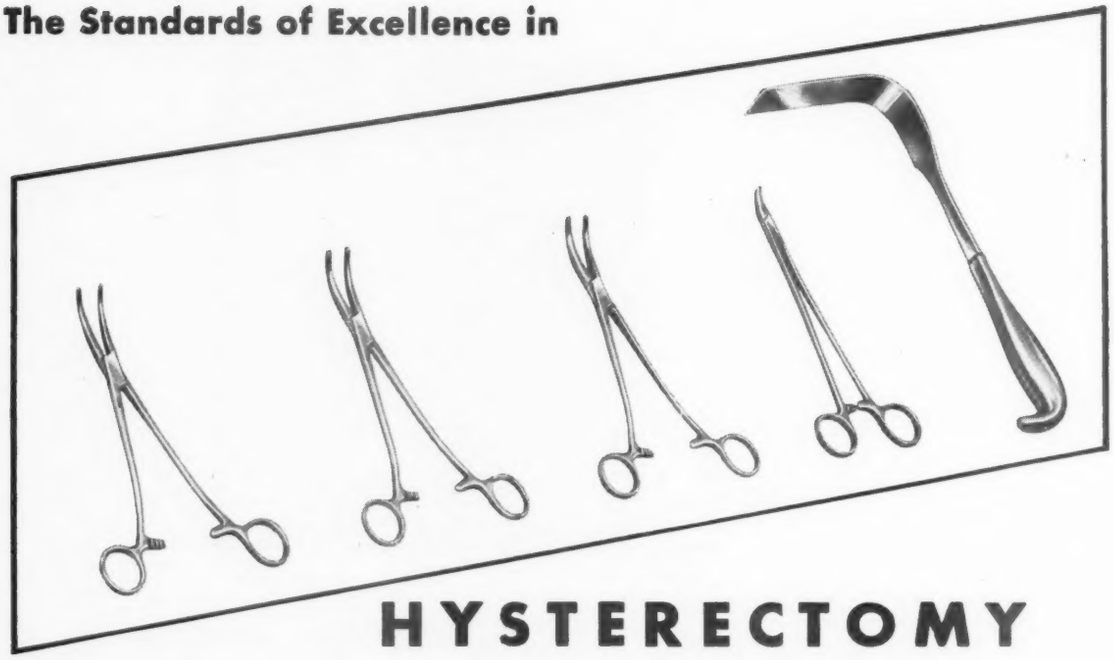
Supply: 50 cc. vials.

SQUIBB

Squibb Quality—the Priceless Ingredient

"RENOGRAFIN"® AND "RETROGRAFIN" ARE SQUIBB TRADEMARKS

The Standards of Excellence in



HYSTERECTOMY INSTRUMENTS

(Illustrated left to right)

GL–S3100 HEANEY Hysterectomy Forceps. Short, full-curved, tapering jaws have serrations cut on the bias, with an additional single wide tooth and groove for secure hold. 8¼ inches. Box lock. Stainless steel. **Each, $17.60**

GL–S3110 HEANEY-KANTER Hysterectomy Forceps. Modified Heaney forceps, with additional wide tooth and groove, preferred by some operators. 8¼ inches. Box lock. Stainless steel. **Each, $19.80**

GL–S3120 HEANEY-BALLENTINE Hysterectomy Forceps. Modified Heaney forceps, with grooves in both jaws running longitudinally, so that tissue must pass through an undulating course, increasing traction and giving a more secure bite. Single wide tooth and groove. 8¼ inches. Box lock. Stainless steel. **Each, $29.85**

GL–S3121 HEANEY-BALLENTINE Hysterectomy Forceps. (Not illustrated.) Similar to GL–S3120, except with straight Ballentine jaws. For abdominal hysterectomy. 8¼ inches. Stainless steel. **Each, $29.85**

SU–S16120 HEANEY Needle Holder. Curved jaws. 8¼ inches. Box lock. Stainless steel. **Each, $17.00**

SU–S16122 HEANEY Needle Holder. As above, but with Ochsner Diamond Jaws. Stainless steel. **Each, $28.50**

GL–S350 HEANEY-SIMON Hysterectomy Retractor. Improved, with blade end slightly curved. 1" x 4½" blade, on 10½" shaft, giving maximum exposure, occupies minimum space. Stainless steel. **Each, $16.00**

V MUELLER & CO.

330 South Honore Street **Chicago 12, Illinois**

Dallas • Houston • Los Angeles • Rochester, Minn.

Armamentarium

now available
the parenteral form

in postoperative states
in hyperemesis of pregnancy
in labor

effective prevention
and rapid control of emesis and anxiety

Trilafon® Injection
perphenazine

full-range tranquilizer—potent antiemetic

greater milligram effectiveness than other parenteral phenothiazines

- virtually no significant hypotension
- no agranulocytosis reported
- no skin photosensitivity observed
- pain on injection absent

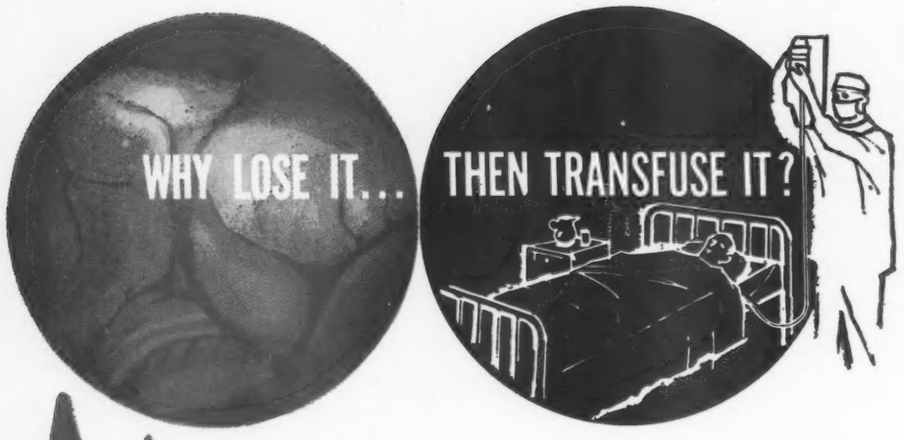
TRILAFON Injection—5 mg., ampul of 1 cc., boxes of 6 and 100.

Refer to Schering literature for specific information regarding indications, dosage, side effects, precautions and contraindications.

TR-J-418

Schering

SCHERING CORPORATION • BLOOMFIELD, NEW JERSEY



REDUCE BLOOD LOSS DURING SURGERY

KUTAPRESSIN®

Kutapressin, a fractional derivative of liver, minimizes capillary oozing and often reduces the need for transfusion.

Surgical procedures, such as tonsillectomies and adenoidectomies, transurethral resections, and even ophthalmologic and brain surgery, are facilitated by improving visibility within the surgical field. The incidence of postoperative hemorrhage is likewise significantly reduced.

In all surgical procedures, Kutapressin promotes better wound healing. Kutapressin does not affect systemic blood pressure, blood clotting or prothrombin time, and there are no untoward effects following its use.

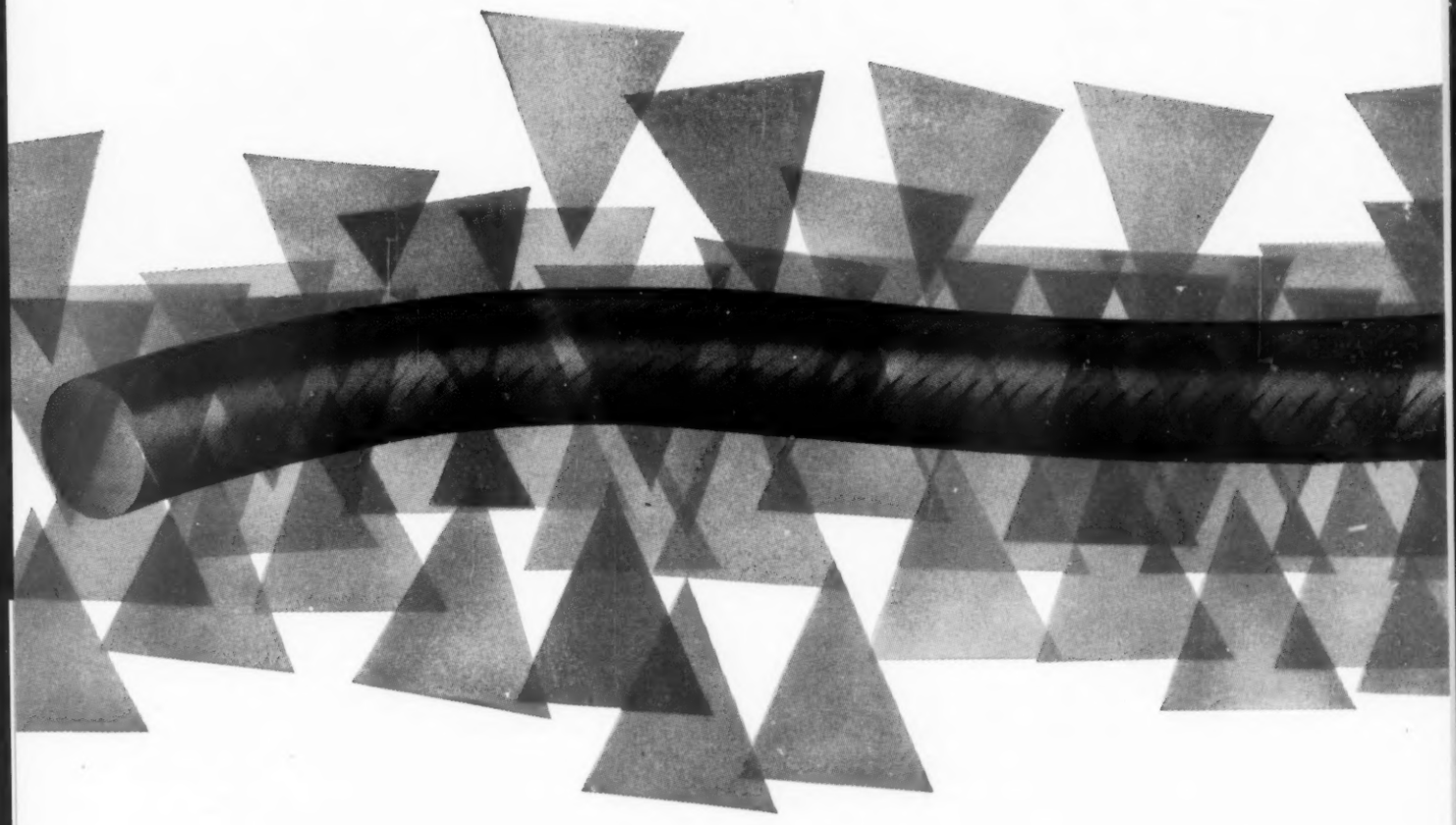
Kutapressin is administered intramuscularly or subcutaneously. Available in 10 cc. and 20 cc. multiple dose vials.

send for reprints and literature

Prescribe with Confidence

KREMERS-URBAN COMPANY • *Milwaukee 1, Wisconsin*

Ethical Pharmaceuticals Since 1894



Electron Sterilized

Surgical Gut...stronger, more pliable

T.M.

This symbol is the trademark of ETHICON, INC. for electron beam sterilization. It identifies Ethicon electron sterilized surgical gut and is your guarantee of maximum strength, pliability and sterility.

Electron sterilized ETHICON Surgical Gut is approximately 10% stronger, more pliable, easier to handle than ordinary surgical gut. High temperatures used in heat sterilization are eliminated—yet the lethal ELECTRON BEAM dose is 40% greater than that required to kill even the most resistant micro-organisms.

ETHICON®

new study confirms
EFFECTIVENESS

101
out of
106

infected wounds
successfully
treated with

Panafil®
OINTMENT

"...offers the surgeon another and more efficient approach to the treatment of infected surface lesions."*

A recent clinical study* has shown PANAFIL Ointment to be effective in 101 out of 106 suppurative wounds and ulcerations involving a variety of bacterial organisms. PANAFIL, the only local medication used in these cases, "...produced uniformly successful results." Systemic antibiotics were employed only in the more severe infections.

Three ingredients in PANAFIL produce a continuous *cleansing-protecting-healing* action in the wound:

Papain —effective debriding enzyme, gently and safely removes debris and slough detrimental to the healing process.

Urea —augments the cleansing action of papain, especially in encrusted lesions.

Chlorophyll derivatives —control inflammation, promote the growth of normal, healthy granulations.

PANAFIL Ointment contains papain 10%, urea U.S.P. 10%, and water-soluble chlorophyll derivatives 0.5% in a hydrophilic ointment base. Available in 1-ounce and 4-ounce tubes *on prescription only*.

Miller, J. M.; Godfrey, G. C.; Ginsberg, M., and Papastrat, C. J., in Surgical Service, V. A. Hospital, Fort Howard, Maryland: Postgrad. Med. *22*:609, 1957.

Rystan company • **Mount Vernon, N. Y.**

29459

UNPARALLELED *stability • sharpness*
strength • malleability • control

DEKNATEL 'K' NEEDLE

PATENT PENDING

...STARTS AS AN EXTREMELY SHARP PENETRATING POINT

...CONTINUES AS A TAPER POINT—NO CUTTING SIDES

PENETRATING POWER IS COMPLETELY CONFINED TO THE POINT

THE EXTREME SHARPNESS AND ORIENTATION OF THE DEKNATEL 'K' NEEDLE PENETRATING POINT AFFORD THESE ADVANTAGES:

- **BENDING OF NEEDLES MINIMIZED OR ELIMINATED.** The point engages the tissue immediately and advancement is made with minimum effort.
- **NEEDLE HOLDER STABILITY IS MAXIMAL.** Since very little force is needed to advance the point, twisting in the holder is decreased or eliminated entirely, no matter at what angle the Deknatel 'K' Needle is held.
- **HEMOSTAT BECOMES A NEEDLE HOLDER WHEN DESIRED.** Because of the ease of passage, the excessive force of needle holder jaws is not necessary to stabilize the Deknatel 'K' Needle. Any ordinary clamping instrument will do.
- **USE OF FINER GAUGE NEEDLES THAN PREVIOUSLY POSSIBLE** because the sharpness carries the finer diameter through without bending.
- **SMALLEST DIAMETER HOLE POSSIBLE.** The Deknatel 'K' Needle point is completely within the diameter of the wire, thus making the hole of a taper point needle—the smallest one possible. There are no lateral projections of cutting sides to enlarge the hole.
- **SEMI-MALLEABILITY.** Curvature of the Deknatel 'K' Needle may be changed at will for a particular modality without increasing the danger of its breaking or bending in use.

ADDITIONAL ADVANTAGES

GENERAL SURGERY

- **FINEST APPROXIMATION OF SKIN WOUNDS.** Because of the orientation of the point, the very edges of skin are coapted with ease, producing superior cosmetic results and decreasing the scarring potential.
- **SKIN CLOSURES WITH FINER GAUGE NEEDLES THAN EVER USED BEFORE.** Closure is readily made with a 6-0 arteriovascular-type needle—a finer diameter than the equivalent ophthalmic needle. The use of finer diameters also decreases the scarring potential.
- **EXTREMELY FINE PICKUP WITHOUT TEARING OR CUTTING OUT OF TISSUE.** Subcuticular Bites: The absence of cutting sides and the orientation of the point allows the pick-up of finest sections with minimal trauma.
- **PASSING OF RETENTION SUTURES.** It is no longer necessary to 'wrestle' a needle through tissue. The extreme sharpness of the Deknatel 'K' Needle gives unusual ease of passage.
- **SAME NEEDLE FOR PERITONEUM, MUSCLE, FASCIA AND SKIN.** The absence of cutting edges on the Deknatel 'K' Needle outmodes the need for changing from taper to cutting edge needles. The Deknatel 'K' Needle is all-purpose in closure.

OB-GYN SURGERY

Mucosal layers are picked up with almost microtome thinness as in the Le Fort operation.

The needle's malleability avoids the danger of its breaking in vaginal plastic repair.

THORACIC SURGERY

- **PENETRATION OF CARTILAGE WITH SMALLEST POSSIBLE HOLE** such as costochondral junctions and trachea. A 6-0 arteriovascular needle with a Deknatel 'K' point easily penetrates the bronchus with greatly decreased probability of splitting.
- **LIGATION OF BLEEDERS ON THE SURFACE OF THE PERIOSTEUM AND TRACHEA.** Fibrous tissue will not be torn. There are no cutting sides, no cutting in or out.
- **PASSAGE THROUGH AN ARTERIOSCLEROTIC PLAQUE.** The Deknatel 'K' Needle is sharp enough and tough enough to aid greatly in the penetration of the calcification.

OPHTHALMIC SURGERY

- **IN CORNEAL AND SCLERAL WORK** the Deknatel 'K' Needle more closely approaches the ideal than any conventional cutting, reverse cutting or Grieshaber-type needles. There are no cutting sides to split fibrous tissue. There are no cutting surfaces above or below to split or cut adjacent structures. The upper flat surface picks up fibrous tissue in microtome thinness as desired.

PLASTIC SURGERY

- **CLEFT PALATE.** The needle curvature may be varied into a 'V' or any other shape. The needle will not break in bending or in use.

OTOLARYNGOLOGIC SURGERY

The Deknatel 'K' Needle may be formed into a 'crochet hook' shape. There is no danger of breaking. The sharpness of the point will carry the needle through the tissue.

For samples, write –

DEKNATEL 96-36 222 STREET, QUEENS VILLAGE 29, N. Y.

WHEN POTENCY COUNTS MOST

Lilly POLIO VACCINE

THERACEBRIN

(Pan-Vitamins, Therapeutic, Lilly)

aids in the rehabilitation of severely ill or injured patients

B04005

Lilly

QUALITY / RESEARCH / INTEGRITY

The American Journal of Surgery

VOLUME 96 JULY 1958 NUMBER ONE

EDITORIAL

The Challenge of Emergency Clinics

It would seem that the time has arrived for all surgeons to reconsider the importance of emergency clinics to patient care, surgical practice and surgical training. If this remains our "weakest link" in hospital care, it is most certainly a link growing in size if not in strength. There continues a rapid increase in patient load in most hospital emergency clinics which apparently began after World War II. Without a study of individual hospitals in conjunction with changes in the local community one can only speculate about the cause of this increase in emergency clinic visits. Perhaps such an evaluation should be made by every hospital with an active emergency clinic.

Shortliffe and associates [1] recently reported the results of a review of emergency clinic facilities in the New England area, and the increase in emergency clinic load over the past ten years is somewhat astounding. It certainly cannot be explained on the basis of population increase alone. A brief review of twenty-five hospitals of 100 or more beds in Ohio shows a similar rise in emergency clinic visits. In 1952, 199,328 patients were seen, whereas in 1956 the number of patients rose to 375,685, an increase of 88.4 per cent. Information is not available as to whether space and personnel have similarly increased. In these same hospitals, however, there has been a 15.4 per cent decrease in the number of outpatient clinic visits despite the fact that six of the hospitals did not have such an organized clinic in 1952 but did in 1956.

What is the cause of this continued growth in our emergency clinics? It would seem that the public is becoming emergency clinic conscious. Facilities are available twenty-four hours a day; there may be a shorter wait than in a physician's office; specialty as well as general medical consultation is easily obtained; many outpatient clinics refuse patients unless they are of a certain income status; some patients have never chosen a family physician; some insurance policies cover emergency clinic care; city emergency squads will transport the sick or injured to a hospital. These are a few of the possible causative factors which bring more patients to our emergency clinics. Each hospital must evaluate its own emergency clinic problems and decide accordingly what must be done.

It seems logical that surgeons should take a most active interest in our growing emergency clinics. Many of these areas are under the direction of the hospital department of surgery and, certainly, over half of the emergency cases seen present surgical problems. The importance of good emergency care cannot be overemphasized. Herein involves an important aspect of doctor-patient relationship which affects public opinion as to the general adequacy of medical care. The young surgeon will find that often the attention given to patients he is called to see in the emergency clinic will affect his whole practice. While the intern continues to be the physician primarily responsible for the immediate care of the emergency patient, more attention must be given this area by residents and staff if a top level of care is to be maintained in this area.

Where hospital residency programs exist, the

[1] SHORTLIFFE, E. C., HAMILTON, T. S. and NOROIAN, E. H. The emergency room and the changing pattern of medical care. *New England J. Med.*, 258: 20, 1958.

emergency clinic is becoming increasingly important. This is the source of many of the cases directly treated by the house staff and from this group will come a larger number of his hospitalized patients. Some figures from a university hospital serve to stress this point further. In a four-year period (1952–1956) the patient load in both the surgical clinics and emergency clinic nearly doubled. Even so, more surgical cases were seen annually in the emergency clinic. In 1956, 27,039 patients, 15,201 of whom presented surgical problems, were examined in the emergency clinic. Almost all these were new patients and therefore required a more extensive evaluation than the revisit cases in the clinics. Furthermore, over 50 per cent of the admissions to the hospital tutorial services, or one of every seven admissions to the hospital (all services), came through the emergency clinic. However, of more importance is the need for surgical residents to give their attention to minor surgical problems. The surgical resident needs to know just as well how to manage correctly simple fractures, lacerations and injuries to the hands as how to perform a cholecystectomy or gastric resection, for this minor surgery will comprise a large part of his later practice.

At first impression it would seem that it might be better not to encourage growth of the emergency clinic but rather to direct more patients to daily outpatient clinics. A review of the cases seen in one emergency clinic, however, shows that less than 10 per cent could be referred directly to an elective clinic, that is, their problems, although not all dire emergencies, presented themselves either when clinics were not open or required earlier management. Increasing emphasis should therefore be given to the emergency service from the standpoint of improving facilities and increasing personnel to improve patient care. In those hospitals with an active teaching program the great variety of clinical material should be utilized to its best advantage.

Each hospital must decide what is the cause of its increased emergency clinic load and what should be done about it. The emergency clinic will probably have to continue to accept all who seek care regardless of income. There may be no way to curtail the rising number of patient visits. Surgeons are more directly involved in this aspect of patient care and can do much to improve not only care but also surgical training in this area. Herein lies a new challenge for all surgeons.

ROGER D. WILLIAMS, M.D.,
Columbus, Ohio

SCIENTIFIC PAPERS

Intrarenal Arteriovenous Fistula with Hypertensive Cardiovascular Disease

REVIEW OF THE LITERATURE

FRANK MILLOY, JR., M.D., EGBERT H. FELL, M.D., ROBERT F. DILLON, M.D.* AND AGENOR M. ZAYAS, M.D., *Chicago, Illinois*

From the Cook County and Presbyterian Hospitals of Chicago; the Hektoen Institute and University of Illinois College of Medicine. Assisted by grants-in-aid from the Chicago Heart Association and the Otho S. A. Sprague Memorial Institute.

AN intrarenal arteriovenous fistula is a unique pathological entity. This lesion is capable of producing not only the physiologic effects of a systemic fistula but also, we believe, those of renal ischemia.

We have been able to find eleven cases of renal arteriovenous fistulas reported in the literature, exclusive of those occurring between renal artery and vein stumps following nephrectomy. Examples of this latter occurrence have also been reported [22] but are not considered here because there is no mechanism by which they might be expected to produce a Goldblatt effect.

Of the eleven cases of renal arteriovenous fistulas previously reported, only two were of traumatic origin. The others were either congenital, due to erosion of the renal vessels by tumor, or of undetermined causes. The infrequency of traumatic arteriovenous fistula of the renal vessels is emphasized by the fact that of the 585 officially recorded cases of arteriovenous fistulas acquired during World War II, none involved the kidney or renal vessels [10].

We are reporting what we believe to be the twelfth case of arteriovenous fistula of the renal vessels and the third of traumatic etiology.

CASE REPORT

On January 15, 1956, a thirty-two year old woman was admitted to Cook County Hospital because of dizziness and headaches of eight years' duration, and ankle edema and easy fatigability of five months' duration. More recently she had been sleeping on three pillows and had also noted vague substernal pain. She had been treated for high blood pressure for six years by private physicians, the last of whom informed us that her blood pressure averaged 180/100 mm. Hg and had been recorded as high as 200/100 mm. Hg. Her past history included a story of having injured herself in the left flank when she fell on a knife in a watermelon patch at the age of ten. At the time of this accident she was taken to a doctor who stopped the bleeding by means of a skin clip.

Physical examination revealed a normally developed, thirty-two year old female, exhibiting mild dyspnea. The temperature was 98.6°F., the pulse rate was 80/minute and the blood pressure was 150/104 mm. Hg. Funduscopy was negative. There were scattered moist rales over both lung fields and the cardiac apex extended to the anterior axillary line by percussion. Auscultation revealed an apical grade II systolic murmur and the second sound was louder over the second left interspace. A gallop rhythm became obvious with exertion. A 1.0 cm. linear scar was observed in the left lateral lumbar region, and in the immediate area a loud continuous bruit could be heard. There

* Deceased.

FIG. 1. Intravenous pyelogram shows distortion of the upper half of the left renal pelvis. The upper calyces are dilated and pushed laterally.

FIG. 2. A plastic cast of the surgical specimen was made by injecting the ureter and renal vessels. The lower half of the kidney is relatively normal. The light-colored pelvis and ureter project from the medial side just below the greatly dilated dark-colored renal vein and aneurysm. The dilated veins had compressed and greatly distorted the upper renal parenchyma.

was a 2 plus pitting ankle edema. Urinalysis revealed a trace of albumin but the sediment was negative for cells or casts. The Kahn test was negative; hematologic work-up was within normal limits and the non-protein nitrogen was 30. The venous pressure was 22 cm. of saline. A roentgenogram revealed that the heart was moderately enlarged in its transverse diameter with diffuse fine nodular densities throughout the lung fields. The arm-to-tongue magnesium sulfate circulation time was normal. Phenolsulfonphthalein excretion was 72 per cent in two hours. An intravenous pyelogram showed specification of the renal pelves of both kidneys at five minutes, with distortion of the left upper pelvis. (Fig. 1.)

TABLE 1
PREOPERATIVE CATHETERIZATION STUDIES

Location	Blood Oxygen Contents (vols. %)		Pressures (mm. Hg)
	Rest	Exercise	Rest
Superior vena cava	10.98	.....	.....
Inferior vena cava, low	11.72	.....	7
Left renal vein	16.09	.....	10
Right atrium, upper	11.81	.....	.....
Right atrium, lower	11.14	.....	7
Pulmonary artery	12.73	12.08	69/32 (40)
Pulmonary "wedge"	.....	.....	23
Brachial artery, content	16.14	16.20	155/69 (102)
Capacity	17.83	.....	.....
Saturation (%)	90.5	90.8	.....
Oxygen consumption/min	288 cc.	418 cc.	.....

	L./min.	Index (L./m.²/min.)	Stroke (cc.)
Cardiac output:			
Rest	8.46	5.13	100
Exercise	10.14	6.15	91
Total pulmonary resistance	378 dynes	Sec./cm.⁵	.....
Pulmonary arteriolar resistance	161 dynes	Sec./cm.⁵	.....

The essential cardiac catheterization findings are given in Table 1. They may be briefly summarized as follows: The patient was in moderate left heart failure as indicated by the elevated pulmonary wedge pressure. The resting systemic venous pressure was within normal limits although a localized elevation was recorded (10 mm. Hg) when the catheter was directed into the left renal vein. A sample of blood taken at this point had an oxygen content almost equal to that of the peripheral arterial blood. The volume of blood shunted through the fistula per minute was calculated to be 2.5 L. or 29.5 per cent of the cardiac output.

Because of the heart failure and evident decreasing cardiac reserve, surgical intervention was elected. The left kidney was explored through

a thoracoabdominal incision extending obliquely upward from the abdominal midline along the ninth interspace. The renal vein was found to be enlarged to three or four times the normal size and it transmitted a prominent thrill. The upper two-thirds of the kidney was enlarged slightly and the parenchyma was compressed by tense dilated veins. The renal artery and vein were dissected as far as possible into the substance of the kidney hilus in the hope that the fistula could be identified and closed without nephrectomy. When it became apparent that the fistula was well within the kidney parenchyma, nephrectomy was performed. A plastic cast of the specimen was made. (Fig. 2.)

The patient had an uncomplicated postoperative course. She no longer has symptoms of cardiac failure. However, her blood pressure has remained moderately elevated at a level of about 160/90 mm. Hg during the first postoperative year.

CLINICAL CLASSIFICATION

We have classified the eleven cases of previously reported renal arteriovenous fistulas into three clinical groups. (Table II.)

The first group consists of those patients who had only hematuria with no evidence of heart failure or hypertension. Only one of these patients had local findings of a murmur or thrill over the lesion. Although two patients had a mildly elevated systolic blood pressure, it was not abnormal for their ages.

The second group consists of those patients with evidence of heart disease but no definite hypertension. Presumably, these patients had sufficiently large shunts to cause cardiac findings, such as any sizable peripheral arteriovenous fistula might, but insufficient alteration in renal hemodynamics to produce a hypertension.

Finally, those patients in whom there is a definite hypertension are placed in the third group. We feel that our case, along with those of Rieder [*30*], Hamilton [*14*], Baron [*4*] and Pelot [*26*] and their respective co-authors fall into this class. Four of the five patients in this group had acquired fistulas, three traumatic and one due to erosion of vessels by a malignant tumor. The two cases of traumatic fistulas are similar to ours and of particular interest, and are reviewed in more detail.

The patient of Pelot et al. [*26*] was a twenty-six year old soldier who was wounded in the right flank in 1951. In 1953 he presented complaints of dyspnea and hemoptysis, findings of an enlarged heart, gallop rhythm, a pulse rate of 110/minute and a blood pressure of 210/130 mm. Hg, in addition to hypertensive retinopathy with papilledema. Retrograde renal catheterization studies revealed distortion of the right renal pelvis. On ureteral catheterization there was a trace of albumin in the urine from the right side and 1 gm./L. of albumin in the urine from the left or uninjured kidney. The fistula was not suspected until surgery, the kidney was removed and that afternoon the blood pressure was 140/80 mm. Hg.

The patient of Baron and Koenemann [*4*] was a seven year old child who presented symptoms of hypertension with a blood pressure of 146/110 mm. Hg eighteen months after sustaining a puncture wound of the abdomen. Her blood pressure five days after the injury had been recorded as 108/60 mm. Hg. She had no eye ground changes and non-protein nitrogen was 27. Nephrectomy was performed and the postoperative blood pressure was 120/80 mm. Hg at the time of discharge from the hospital.

The case we have presented is in many ways similar to these two traumatic cases. The main differences lie in the length of time between the injury causing the fistula and the subsequent nephrectomy, and also the resulting clinical course of the patients. Whereas the previous two patients had nephrectomy less than two years after formation of the fistula, twenty-two years elapsed between injury to the kidney and surgery in our patient. Although our patient was cured of her fistula, she has subsequently been found to have retained an element of hypertension in contradistinction to the previous two patients who were cured of their hypertension.

COMMENTS

Arteriovenous Fistula. Systemic arteriovenous fistulas fall into two etiologic categories: acquired and congenital. The congenital group includes cirsoid aneurysms, pulsating hemangiomas and similar lesions. Acquired fistulas are most often single communications between the large arteries and veins while congenital arteriovenous fistulas are most frequently multiple and involve smaller vessels.

The physiologic effects of an arteriovenous fistula may be considered from two aspects. The first concerns the central effects of the lesion on the systemic circulation as measured by pulse rate, blood pressure and other ap-

TABLE II

PERTINENT DATA ON ARTERIOVENOUS FISTULAS OF RENAL VESSELS REPORTED IN LITERATURE TO THIS TIME*

Author, Date	Age (yr.), Sex	Etiology	Clinical Findings	Blood Pressure (preoperative)	Blood Pressure (postoperative)	Follow-up and Remarks
			Group One†			
Pearse and MacMillan, 1947	60, F	Congenital	Hematuria, no murmur	160/60	.....	Uneventful recovery after surgery
Schulze-Bergman, 1954	59, F	Congenital	Hematuria, no murmur	150/90	.....	Diagnosis made by aortogram; nephrectomy
Myrhe, 1956	66, F	Carcinoma of kidney	Massive hematuria; bruit present	140/70	.....	Cardiac catheterization showed high cardiac output; diagnosis of carcinoma made at surgery
			Group Two‡			
Varela, 1923	28, F	Congenital	Cardiac decompensation; bruit over lesion	160/80	.....	Diagnosis confirmed at postmortem examination
Kirbey et al., 1955	22, M	Congenital or traumatic	30% cardiac enlargement; exertional dyspnea; lumbar murmur and thrill	160/60	Systolic, 120 to 130	Nephrectomy
Slominski-Laws et al., 1956	19, F	.....	Mild cardiac decompensation; lumbar murmur and thrill	120/80	.....	Ankle edema for 3 yr. postnephrectomy
			Group Three§			
Rieder, 1942	....	Congenital	Cardiac decompensation; thrill and murmur present	220/120	.....	"Cured by nephrectomy"
Hamilton et al., 1953	29, M	Renal carcinoma	Mild cardiac decompensation; bruit present	180/110	140/90	.....
Baron and Koenemann, 1955	7, F	Traumatic	Constant severe headache; lumbar thrill and bruit	146/110	120/80	An early blood pressure had been recorded at 108/60
Pelot et al., 1954	26, M	Traumatic	"Malignant hypertension"	210/130	135/75	Evidently cured by nephrectomy
Case presented herein	32, F	Traumatic	Hypertension; mild cardiac decompensation	180/100	Variable, up to 160/90	Hypertension remained after nephrectomy

* One case is not included because of lack of information.

† Patients in whom the primary symptoms and findings were due to the local effects of the lesion, principally hematuria.

‡ Patients with cardiac symptoms or findings as a result of the fistula, but without hypertension.

§ Patients with hypertension.

propriate determinations; the second concerns the effect of the lesion upon the circulation to the tissues distal to the fistula.

The central physiologic effects of a systemic arteriovenous fistula other than those of one involving the renal vessels have been clearly defined by Holman [*7,16*] and others [*2,29*]. Although these effects are usually more pronounced in acquired lesions, they are frequently present or will develop eventually in congenital lesions. Adams [*1*], in a review of twenty-two congenital arteriovenous fistulas, stated that these lesions progress slowly but eventually show all of the classic findings, with a local thrill and bruit, and finally cardiac hypertrophy and decompensation.

The surgical creation of an arteriovenous fistula of adequate caliber between a peripheral artery and vein is immediately followed by a fall in systolic and diastolic blood pressure, with a gradual recovery of the systolic pressure. The resumption of the pretraumatic systolic pressure is thought to be due to compensatory mechanisms such as increased blood volume and increased cardiac output. The diastolic pressure, however, remains low, resulting in an increased pulse pressure.

That peripheral arteriovenous fistulas other than those of the renal vessels do not cause significant elevation of systolic pressure has recently been confirmed by Epstein, Post and McDowell [*11*]. They studied seventeen veterans of the Korean conflict with traumatic arteriovenous fistulas of the extremities and found a resting systolic pressure of less than 120 mm. Hg in thirteen of the seventeen. The highest recorded resting systolic pressure in their seventeen patients was 134 mm. Hg.

Although these systemic effects of arteriovenous fistulas are well known and can be easily measured, the effects of an arteriovenous fistula on the tissues distal to it are not so easily predicted or determined. A few general statements may be made. In the case of acquired fistulas there is increased circulation in the immediate vicinity of the lesion [*29*]. This is due to the great increase in collateral circulation around the fistula. However, clinical and experimental evidence indicates that a frequent consequence on distal tissues supplied by an artery involved in arteriovenous fistula is ischemia. This is due to the short-circuiting effect of the lesion, which causes much of the blood to follow the path of lesser resistance through the fistula rather than its normal course through the peripheral capillary bed [*7*]. Schumacker [*8*] states that there is commonly evidence of increased circulation distal to an arteriovenous fistula but that sometimes there is coldness, pallor and weaker pulses with reduced blood pressure in an extremity as a result of this lesion. Reid [*29*] states that there is abundant clinical and experimental evidence which indicates that an arteriovenous fistula not only does not improve the circulation to the part beyond it but also, on the contrary, actually embarrasses it. He states further that in patients the frequent occurrence of cyanosis, lowered surface temperature, edema and ulceration, and the occasional presence of intermittent claudication as a result of a fistula between the large arteries and veins speak strongly for the deleterious effects of a fistula on the circulation of that part supplied by the vessel involved.

Holman and Taylor [*17*] demonstrated experimentally that the effect of a fistula depends upon the relationship of its size to the caliber of the vessels in which it lies. By pressure studies and a dye injection technic they showed that if a fistula is large enough, the flow in the distal artery is reversed, so that blood flows retrograde from the artery through the fistula into the vein. In four dogs in which they constructed iliac fistulas and measured pressures in the proximal and distal portions of the artery, they noted a drop of systolic and diastolic pressures of as much as 30 to 40 mm. Hg distal to the artificial fistula.

In another experimental study, Robertson, Dennis and Elkin [*31*] took direct measurements of extremity blood flow by the plethysmographic method of Eckstein. They found that in an extremity with an arteriovenous fistula the flow values with the fistula open may be considerably lower than the average normal values, even though the viability of the limb as well as a moderate tolerance for exercise would appear to indicate that the nutrition of the tissue is adequate.

Although congenital arteriovenous fistulas are frequently characterized by an increase in skin temperature and hypertrophy of the involved part, which may be interpreted to indicate an increase in blood supply, there is likewise evidence for a decrease of the blood supply to peripheral tissues in lesions of this type. In a series of nine cases of congenital fistulas reported by Pemberton and Saint [*27*], all four

cases with extremity involvement were prominently characterized by ulceration and gangrene. They concluded that the local regional effects of acquired and congenital arteriovenous fistulas were similar.

Another effect which might be expected in the vascular tree distal to a peripheral arteriovenous fistula is a lowering of the arterial pulse pressure. Although there is an increased pulse pressure in the general circulation of a patient with an arteriovenous fistula, there is no assurance that this would prevail in the artery distal to an arteriovenous fistula. Holman and Taylor [*17*] made observations in five animals in which they had constructed fistulas. They took direct arterial pressures and found that the pulse pressure distal to the fistula was constantly lower than that proximal, being reduced from 62 mm. Hg to 32 mm. Hg and from 30 mm. Hg to 12 mm. Hg in the two most marked examples.

We could find no clinical observation or experimental evidence pertaining to arteriovenous fistulas in visceral organs comparable to that which can be found dealing with this lesion in extremities. However, it is seemingly possible that the renal parenchyma distal to an arteriovenous fistula might become ischemic, and it is also likely that the lowered systolic and diastolic blood pressures and lowered pulse pressures observed experimentally by Holman and Taylor [*17*] would prevail.

Goldblatt Effect. The phenomenon of creating hypertension by partial constriction of the renal artery as described by Goldblatt [*12*] is well known. To summarize, in an animal in which hypertension has been induced by partial constriction of the renal artery, excision of this "Goldblatt kidney" at the height of the hypertension results in a prompt return to normal of the pressure [*32*]. This phenomenon is explained on a humoral basis. The present theory [*12*] is that renal artery constriction causes the kidney to produce renin, which reacts with a plasma constituent, hypertensinogen, to release an active pressor substance, angiotonin or hypertensin. Removal of the kidney in the early stages of this condition lowers the blood pressure, presumably by abolishing the production of renin.

There are numerous reports in the literature [*6,32,33,35*] of unilateral lesions of the renal artery causing malignant hypertension, thus giving clinical support to the experimental observation of Goldblatt. Bauer and Forbes [*5*] in 1952 presented a patient who came to autopsy with malignant hypertension; one renal artery was partially obstructed by a plaque. Whereas this kidney was relatively normal, the opposite kidney, with a normal renal artery, showed arteriosclerosis with necrotizing arteriolitis. They refer to several similar cases which have been reported and attribute the pathologic changes in the kidney with a normal renal artery to a Goldblatt effect caused by the kidney with the partially occluded renal artery.

Trueta and associates [*38*], in an exhaustive investigation of the renal circulation, have found good evidence of a secondary "short-circuiting" system of arteries which are thought to be capable of shunting the blood flow past the renal cortex. They postulate that excessive diversion of the intrarenal blood flow from the cortex through this medullary bypass may, by the Goldblatt mechanism, be a factor in the production of human hypertension. If one were to carry this reasoning one step further and place the "short circuit" at the level of larger renal arteries and veins in the form of an arteriovenous fistula, the same result, namely, a Goldblatt hypertension, should be expected.

Although there is still controversy regarding the exact mechanism of the production of renin, three of the factors which have been suggested as causative are: (1) renal ischemia, or a decrease in total blood flow; (2) decreased pulse pressure in the renal artery without an absolute decrease in renal blood flow [*19*]; and (3) a decrease in the intrarenal volume pulsation [*15*]. Since two of these effects, decreased pulse pressure and tissue ischemia, have been demonstrated both clinically and experimentally distal to an arteriovenous fistula, there is at least a theoretical basis for suggesting the possibility of the occurrence of the Goldblatt phenomenon as a result of an arteriovenous fistula proximal to or within the renal parenchyma.

Persistent Hypertension Following Nephrectomy. It has been noted that after constriction of the renal artery in the Goldblatt manner, renin concentration in the blood increases for some weeks but ultimately disappears, even though hypertension persists. It is thought by many investigators that some organic mechanism is operative after the original humoral induction of hypertension by renin.

There is general agreement in the literature

that nephrectomy for unilateral kidney disease causing hypertension gives only fair results [*13,35*]. Thompson and Smithwick [*37*], reviewing the literature in 1952, found reports of about 300 cases and believed that only 24 per cent could be classed as cures. On a theoretical basis this may be accounted for by the fact that after a certain period of the Goldblatt effect, degenerative vascular changes are responsible for hypertension replacing the humoral factors which originally operated. Thus a patient treated by nephrectomy after these vascular changes had taken place would not be immediately benefited, and would only be benefited if the changes were reversible. Presumably, the twenty-two–year duration of the fistula explains why the patient we have presented manifests persistent hypertension after nephrectomy. Whether the vascular changes which had developed in her remaining kidney are reversible remains to be seen.

Experimental Association of Arteriovenous Fistula and Goldblatt Kidney. So far, the numerous systemic and local effects which an arteriovenous fistula and a Goldblatt kidney exert independently have been considered. We would now like to review briefly some of the experimental work which has been carried out in an effort to determine the end effect of these two lesions operating simultaneously.

Lasker and Glenn [*20*], recognizing the fact that an arteriovenous fistula between the renal artery and vein would be a unique lesion from the standpoint of pathologic physiology, created this lesion in a series of dogs. They observed only a mild transient hypertension and postulated that the altered pressure in the renal vein may have altered any Goldblatt effect.

Investigations along a different line were carried out by Deterling and Essex [*9*]. Since a decreased renal artery pulse pressure has been advanced as one of the causes of the Goldblatt effect [*19*], they theorized that a peripheral arteriovenous fistula might counteract the Goldblatt effect by elevating the systemic pulse pressure. They produced hypertension in a group of dogs by the Page technic [*24*], in which the kidneys are wrapped with cellophane and hypertensive levels achieved by constrictive perinephritis. They then created femoral arteriovenous fistulas in the animals. They found that this did not have an antihypertensive effect, even though the arterial pulse pressure was increased. The mortality among their dogs with both hypertension and a peripheral arteriovenous fistula was higher than mortality in dogs with only one of the two lesions.

A third group of investigators, Hawthorne, Brownlee and Pague [*15*], theorized that although the hypertension associated with perinephritis (such as was produced by Deterling and Essex) might not be counteracted by a peripheral arteriovenous fistula, hypertension associated with narrowing of the renal artery might be altered by the production of this lesion. They induced experimental hypertension by means of renal artery constriction rather than perinephritis. They then found that subsequent creation of a femoral arteriovenous fistula caused the blood pressure to return to normal. They suggested that the hypertension caused by both partial renal artery constriction and wrapping of the kidney was due more to a decrease in intrarenal volume pulsation than to actual renal ischemia. They further reasoned that a peripheral arteriovenous fistula, by increasing the systemic pulse pressure would not increase the renal volume pulsation in a wrapped kidney with perinephritis. However, in animals with hypertension resulting from renal artery constriction they expected to increase renal volume pulsation by experimentally producing an increased pulse pressure. Their experiments, in their opinion, tended to confirm this theory.

Since Holman and Taylor [*17*] have demonstrated that an arteriovenous fistula diminishes the distal arterial pulse pressure, it appears that the findings of Hawthorne, Brownlee and Pague would not be applicable to the hypertension caused by a Goldblatt effect resulting from an arteriovenous fistula of the renal pedicle. An arteriovenous fistula of the renal pedicle supposedly would decrease the pulse pressure in the kidney distal to itself, thereby nullifying the effect of the elevated pulse pressure in the remainder of the systemic circulation.

CONCLUSIONS

Arteriovenous fistulas in the general circulation do not as a rule cause significant arterial hypertension [*11*], but are commonly associated with a lowered diastolic blood pressure. Arteriovenous fistulas of the renal vessels may produce

significant arterial hypertension with elevation of both the systolic and diastolic components.

There is experimental evidence that in the tissues distal to an arteriovenous fistula there is a decreased total blood flow [*31*], decreased systolic and diastolic blood pressure, and decreased pulse pressure [*17*].

The majority of acquired arteriovenous fistulas studied in patients have been in the extremities. Although there is an increased circulation in the immediate vicinity of the fistula due to the increased volume of blood carried in collateral channels, most investigators agree that the distal tissues are frequently ischemic, due to the blood-shunting effect of these lesions. Weak pulses, pallor, coolness, pain and gangrene have been associated with this lesion in extremities. Less is known about the status of distal tissues in congenital arteriovenous fistula. Although this condition is frequently associated with hypertrophy of the involved part, which would lead to the conclusion that the circulation is improved, it has likewise been associated with atrophy [*29*] and some authorities [*27,29*] believe that the effects of the two types of fistula on distal tissues is the same, namely, that there is ischemia.

The cause and effect relationship of renal artery constriction and arterial hypertension first demonstrated by Goldblatt has been well established experimentally and clinically. There has been controversy as to whether the Goldblatt effect is caused by renal ischemia or decreased renal artery pulse pressure. Both of these conditions may exist in tissues beyond an arteriovenous fistula of the renal vessels.

Of the eleven cases of renal arteriovenous fistula which we have found reported in the literature, and with the addition of the case reported herein, five patients have had marked hypertension. In one of these patients the etiology was a congenital arteriovenous fistula, in one it was due to erosion of the renal artery and vein by neoplasm, and in three (including ours) it was traumatic.

We believe that the hypertension associated with renal arteriovenous fistula, particularly of acquired etiology, may be on the basis of a Goldblatt effect resulting from the altered renal hemodynamics produced by the fistula.

SUMMARY

Arteriovenous fistulas of the renal vessels are of particular interest because of the ability of these lesions to produce not only the systemic effects of an arteriovenous fistula but also, theoretically, those of a Goldblatt kidney.

Intrarenal arteriovenous fistula is a rare lesion. We have been able to find only eleven cases reported in the literature; six of these were congenital, four were acquired and one was of undetermined etiology.

We believe our case is the twelfth recorded of intrarenal arteriovenous fistula and the third of traumatic etiology. Although arteriovenous fistulas of other systemic vessels do not produce a significant hypertension, those of the renal vessels may.

Clinical opinion indicates that there may be ischemia distal to an arteriovenous fistula, and experimental evidence indicates that there is ischemia and decreased pulse pressure distal to an arteriovenous fistula.

The Goldblatt phenomenon of producing hypertension by partial occlusion of the renal artery is well known.

We believe that the hypertension associated with certain renal arteriovenous fistulas may be explained on the basis of the Goldblatt phenomenon resulting from the altered renal hemodynamics produced by the fistula.

REFERENCES

1. Adams, H. D. Congenital arteriovenous and cirsoid aneurysms. *Surg., Gynec. & Obst.*, 92: 663, 1951.
2. Allen, E. V., Barker, N. W. and Hines, E. A. Peripheral Vascular Disease. Philadelphia, 1955. W. B. Saunders Co.
3. Barker, N. W. Hypertension and unilateral renal disease. *M. Clin. North America*, 35: 1041, 1951.
4. Baron, G. J. and Koenemann, R. H. Arteriovenous fistula of the renal vessels. *Radiology*, 64: 85, 1955.
5. Bauer, H. and Forbes, G. L. Unilateral renal artery obstruction associated with malignant nephrosclerosis confined to the opposite kidney. *Am. Heart J.*, 44: 634, 1952.
6. Blackmen, S. S., Jr. Arteriosclerosis and partial obstruction of the main renal arteries in association with "essential" hypertension in man. *Bull. Johns Hopkins Hosp.*, 65: 353, 1939.
7. Christopher, F. A Text Book of Surgery, 5th ed. Section by E. Holman. Philadelphia, 1949. W. B. Saunders Co.
8. Davis, L. E. Christopher's Text Book of Surgery, 6th ed. Section by H. B. Schumaker. Philadelphia, 1956. W. B. Saunders Co.
9. Deterling, R. A. and Essex, H. E. Experimental renal hypertension and its course following the production of a peripheral arteriovenous fistula. *Ann. Surg.*, 132: 129, 1950.
10. Elkin, D. C. and DeBakey, M. E. Vascular surgery in World War II. U. S. Government Printing Office, Washington, 1955.

11. Epstein, F. H., Post, R. S. and McDowell, M. The effect of an arteriovenous fistula on renal hemodynamics and electrolyte excretion. *J. Clin. Investigation*, 32: 233, 1953.
12. Goldblatt, H. The Renal Origin of Hypertension. Springfield, 1948. Charles C Thomas.
13. Grollman, A. Conference on factors regulating blood pressure. Transactions, Josiah Macy, Jr. Foundation, p. 27, 1947.
14. Hamilton, G. R., Getz, R. J. and Shephard, J. Arteriovenous fistula of the renal vessels, a case report and review of the literature. *J. Urol.*, 67: 203, 1953.
15. Hawthorne, E. W., Brownlee, G. V. and Pague, W. G. Arterial pressure changes in experimental renal hypertensive dogs following production of femoral arteriovenous fistula. *Am. J. Physiol.*, 180: 65, 1955.
16. Holman, E. Arteriovenous Aneurysm. New York, 1937. Macmillan Co.
17. Holman, E. and Taylor, G. Problems in the dynamics of blood flow. II. Pressure relations at the site of an arteriovenous fistula. *Angiology*, 3: 415, 1952.
18. Kirbey, C. K., Nichols, W. G., Garitona, H. P., Wohl, G. T. and Pietroluongo, A. L. Arteriovenous fistula of the renal vessels, a case report. *Surgery*, 37: 267, 1955.
19. Kohlstaedt, D. G. and Page, I. H. The liberation of renin by perfusion of kidneys following reduction of pulse pressure. *J. Exper. Med.*, 72: 201, 1940.
20. Lasker, E. P. and Glenn, F. Effect on kidney and blood pressure of artificial communication between the renal artery and vein. *Arch. Surg.*, 38: 886, 1937.
21. Lewis, T. Vascular Disorders of the Limb. London, 1949. Macmillan Co.
22. Muller, W. H. and Willard, G. E. Renal arteriovenous fistula following nephrectomy. *Ann. Surg.*, 144: 240, 1956.
23. Myhre, J. Arteriovenous fistula of the renal vessels, a case report. *Circulation*, 14: 185, 1956.
24. Page, I. H. Production of persistant arterial hypertension by cellophane perinephritis. *J. A. M. A.*, 113: 2046, 1939.
25. Pearse, R. and MacMillan, R. L. Congenital aneurysm of the renal artery. *J. Urol.*, 51: 235, 1947.
26. Pelot, G., Pessereau, G. and Daftari, B. Hypertensions malignes et nephropathies unilaterales. À propos de 4 observations. *J. urol.*, Paris, 60: 245, 1954.
27. Pemberton, J. de J. and Saint, J. H. Congenital arteriovenous communications. *Surg., Gynec. & Obst.*, 46: 470, 1928.
28. Pemberton, J. de J. and Waugh, J. Traumatic arteriovenous aneurysm; report of eleven cases. *S. Clin. North America*, 19: 981, 1939.
29. Reid, M. Abnormal arteriovenous communications, acquired and congenital. III. Effects on heart, blood vessels and other structures. *Arch. Surg.*, 11: 25, 1925.
30. Rieder, W. Sonderstellung arterio-venoser Aneurysmen der Nierengefasse in Rahmen operativer Behandlung schwerer Herz-Kreislaufschaden beim arterio-venosen Aneurysma. *Chirurg*, 14: 609, 1942.
31. Robertson, R. L., Dennis, I. W. and Elkin, D. C. Collateral circulation in the presence of experimental arteriovenous fistula. Determination by direct measurement of extremity blood flow. *Surgery*, 27: 1, 1950.
32. Sabin, H. S. Hypertension in unilateral renal disease. *J. Urol.*, 59: 8, 1948.
33. Schroeder, H. A. and Fish, G. W. Studies on essential hypertension. III. The effect of nephrectomy upon hypertension associated with organic renal disease. *Am. J. M. Sc.*, 199: 601, 1940.
34. Schulze-Bergmann, G. Über das arterio-venose Aneurysm der Niere. *Ztschr. Urol.*, 47: 661, 1954.
35. Sensenbach, W. S. Effects of unilateral nephrectomy in treatment of hypertension. An evaluation. *Arch. Int. Med.*, 73: 123, 1944.
36. Slominski-Laws, M. D., Kiefer, J. H. and Vermeulen, C. W. Arteriovenous aneurysm of the kidney: case report. *J. Urol.*, 75: 586, 1956.
37. Thompson, J. E. and Smithwick, R. H. Human hypertension due to unilateral renal disease with special reference to renal artery lesions. *Angiology*, 3: 493, 1952.
38. Trueta, J., Barclay, A. E. Daniel, P. M., Franklin, K. J. and Prichard, M. M. L. Studies of the renal circulation. Springfield, 1947. Charles C Thomas.
39. Varela, M. E. Aneurisma arteriovenosa de los vasos renales y asistolia consecutiva. *Rev. med. Latino-Am.*, 14: 3244, 1923.

Studies on Intra-arterial Heparinization

PAUL F. GRYSKA, M.D., *Boston, Massachusetts,* JACK P. ABBOTT, M.D., *Houston, Texas,* AND MICHAEL E. DEBAKEY, M.D., *Houston, Texas*

From the Cora and Webb Mading Department of Surgery, Baylor University College of Medicine, Houston, Texas. Supported in part by Massachusetts Heart Association.

ONE of the many technical aids in successful arterial surgery is the use of local and/or systemic heparinization. Clotting in vessels distal to an area of temporary arterial occlusion is a real hazard seen not infrequently, particularly in operations for obliterative vascular disease. Furthermore, the use of "regional heparinization" [1] is appealing in the postoperative period. This latter technic, however, has not received general acceptance. It would be ideal indeed if both operative and postoperative anticoagulation could be accomplished without a systemic or generalized effect.

To determine the possibility of a localized or regional effect of heparin on coagulation of whole blood, the following study was carried out.

MATERIALS AND METHODS

Adult mongrel dogs were used in this study. All procedures were carried out under pentobarbital anesthesia (30 mg./kg. body weight). Coagulation times were all determined by a standard modified Lee-White technic using three saline-rinsed test tubes (13 by 100 mm.) for each determination and were done at room temperature. All samples from femoral vessels and aorta were obtained by direct puncture of the exposed vessels with dried, clean syringes and No. 20 gauge needles. All iliac artery specimens were obtained via a polyethylene cannula previously placed in the artery.

Five series of experiments were carried out. A control sample for coagulation time was done on each dog prior to each study.

Series I. The distal aorta, right iliac artery and femoral vessels on the right were isolated. The external iliac was ligated flush with the aorta and the distal segment was cannulated with two polyethylene tubes. One tube was used for injection of heparin and the other placed about 1.5 cm. distally for withdrawal of samples. Samples were also obtained after injection from the ipsilateral femoral artery, femoral vein and from the aorta. After control coagulation times were obtained, heparin (0.2 mg./kg. body weight) was injected into the proximally occluded iliac artery cannula. Specimens for effect were drawn at five-minute intervals from aorta, iliac artery, femoral artery and femoral vein. In this experiment heparin in various dilutions from 1 to 80 cc. saline was used (twelve dogs).

Series II. Injection of heparin (0.2 mg./kg. body weight) in 20 cc. saline into flowing iliac artery followed by immediate occlusion of the artery proximal to the site of injection (three dogs.)

Series III. With the right external iliac artery occluded at its origin, heparin (0.2 mg./kg. body weight) in 20 cc. saline was injected intravenously (into inferior vena cava) (four dogs).

Series IV. In this series a continuous, constant perfusion of dilute (4 mg./100 cc. saline) heparin solution into the proximally occluded external iliac artery was carried out for one hour. The injection was maintained at a constant rate by means of an automatic "pipetting machine." Coagulation times were drawn at ten-minute intervals, beginning ten minutes after the start of the perfusion. Specimens taken were from the ipsilateral femoral artery, femoral vein and from the contralateral femoral vein for testing local and systemic anticoagulant response. The ipsilateral femoral arterial specimens were drawn at an average of 5 cm. from the tip of the iliac artery cannula (eight dogs.)

Series V. A continuous, constant perfusion of dilute (4 mg./100 cc. saline) heparin solu-

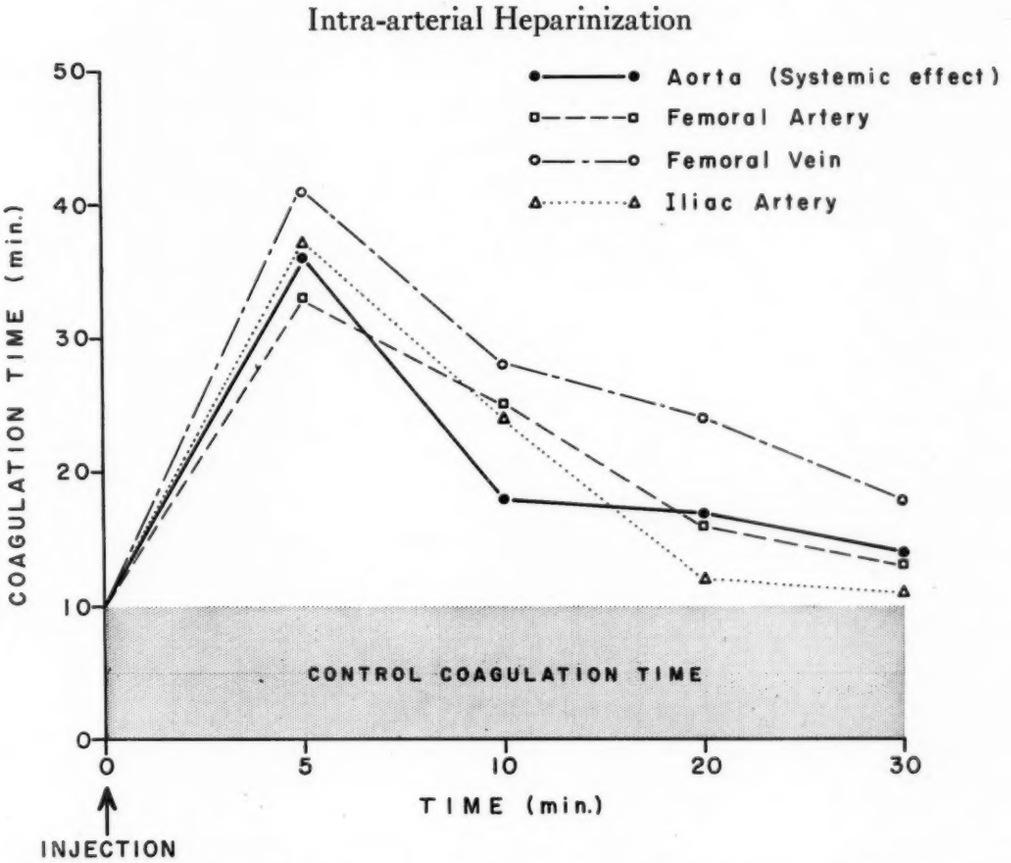


FIG. 1. Single injection of heparin (0.2 mg./kg.) into proximally occluded external iliac artery (twelve dogs).

tion into a flowing external iliac artery was carried out for one hour. A polyethylene cannula was directed through a short incision in the distal aorta into the external iliac orifice for 1 to 2 cm. All specimens were drawn as in series IV (five dogs).

RESULTS

In all the experiments of series I and II, using a single dose of heparin intra-arterially, an immediate generalized anticoagulant response was seen. (Figs. 1 and 2.) A localized response could not be demonstrated. The effect with these small doses (although distinct) was short lived, rarely lasting more than fifteen minutes.

It is interesting that the same effect could be obtained using a single intravenous injection (Fig. 3) even in the proximally occluded iliac artery. This is significant clinically since, if heparinization is deemed worthwhile during surgery, the trauma of multiple arterial punctures in a diseased vessel could be avoided.

In series IV a localized effect was demonstrable only for a relatively short period of time in the proximally occluded external iliac and femoral arteries. (Fig. 4.) In all these studies (save two) the systemic effect of heparin could be seen to approach that of the local effect well before the end of the hour of perfusion. Certainly, whenever a distinct local anticoagulant response was obtained, the effect, in time became generalized. In series V, in contrast to the study of Wylie et al. [1], the effect of continuous, constant intra-arterial heparin perfusion into a flowing vessel was always generalized and relatively equal locally and generally. (Fig. 5.) We were unable to effect "regional heparinization."

In a small series of clinical operative cases, coagulation time studies were carried out after intra-aortic injection of heparin (10 mg. in 10 cc. saline). The aorta was clamped proximally and distally immediately after the injection. Specimens for coagulation time were drawn from sites above and below the clamps at intervals thereafter.

From Figures 6 and 7 it is apparent that the anticoagulant effect of the heparin is rapidly

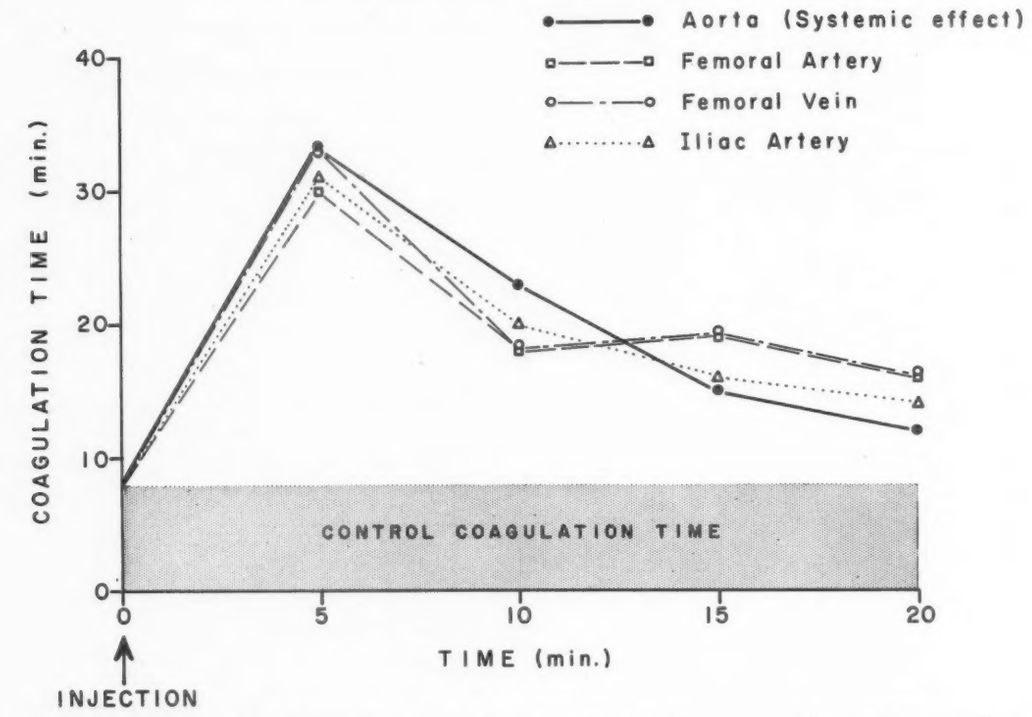


FIG. 2. Single injection of heparin (0.2 mg./kg.) into flowing iliac artery followed by immediate proximal occlusion (three dogs).

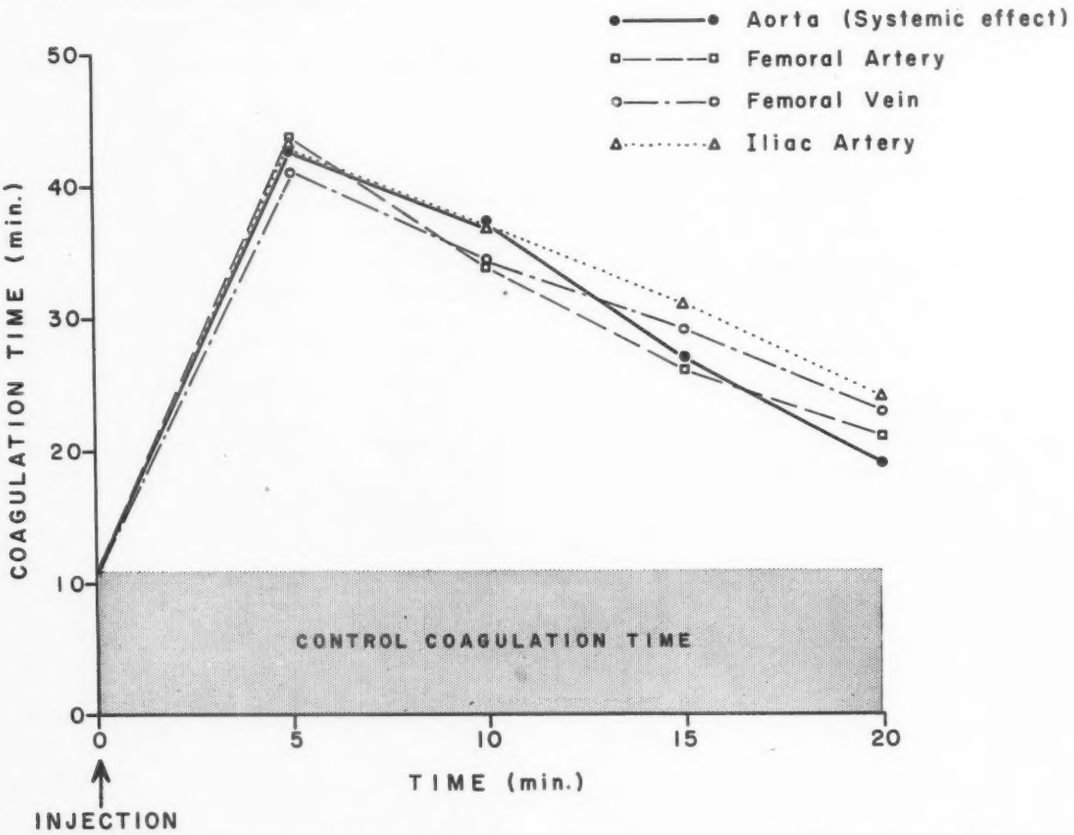


FIG. 3. Single injection of heparin (0.2 mg./kg.) into inferior vena cava with occluded right iliac artery (four dogs).

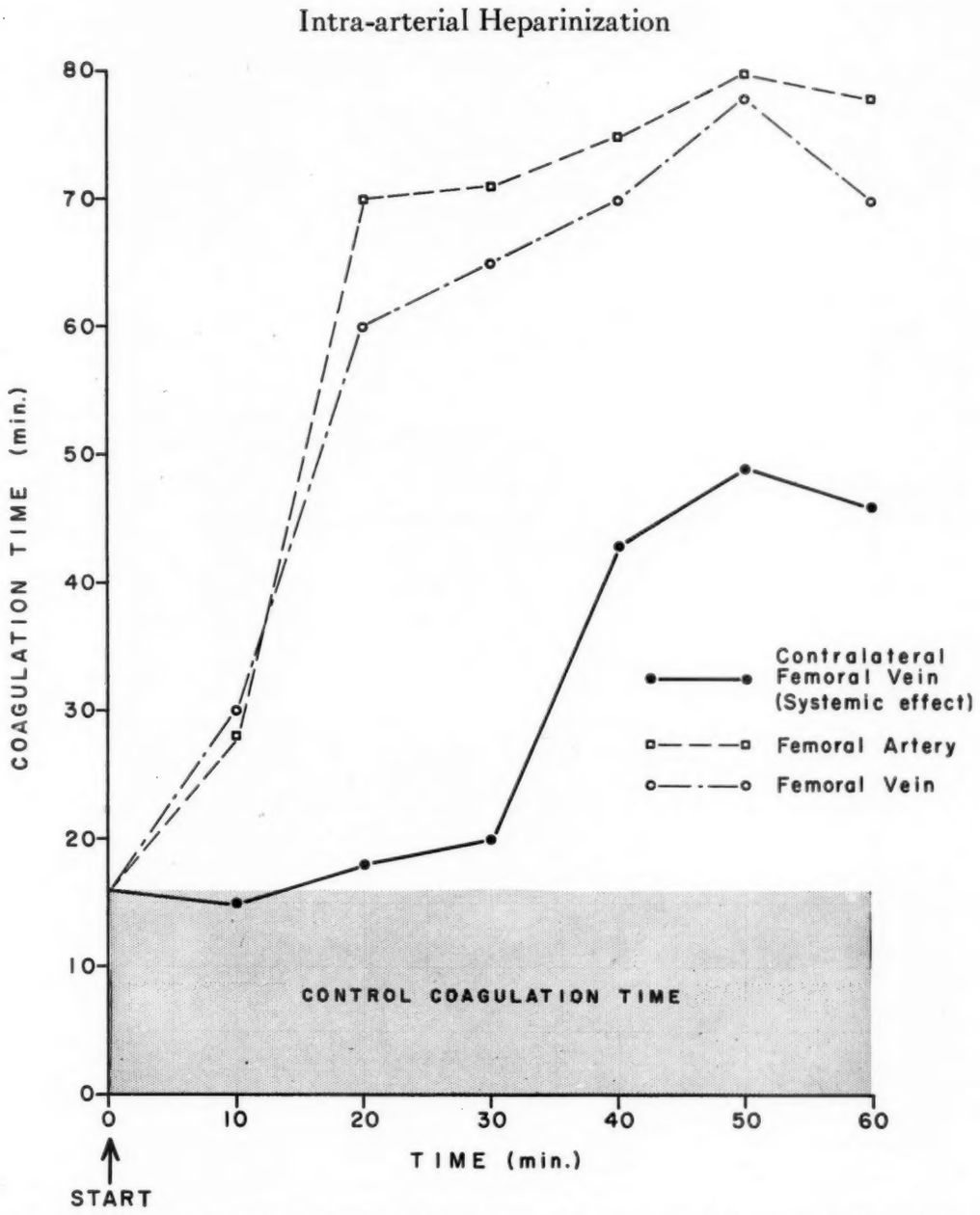


FIG. 4. Continuous injection (by pump) of heparin (4 mg./100 cc. saline) into proximally occluded external iliac artery at 2.3 cc./min. (eight dogs).

evident distal to the site of the injection but is rapidly dissipated. There is a minimal generalized response to the heparin in this dose.

COMMENTS

It would appear that heparin given intra-arterially must be considered as a systemic anticoagulant. The results of this study do not suggest a ready means of trapping the agent effectively to achieve a highly desirable localized effect. It was indeed hoped (in series I and II) that varying the dilution of the agent would permit a localized effect with absence of a systemic one by systemic dilutional means. That this could not be obtained by a single injection or by constant perfusion could, of course, be related to the profuse collateral circulation in the dog. However, its effect in humans at time of surgery is probably similar.

We were frankly disappointed at the inability to obtain "regional heparinzation" in a flowing vessel (series V) as Wylie et al. apparently did. Perhaps in a very low dosage and minimal anticoagulant response this is possible.

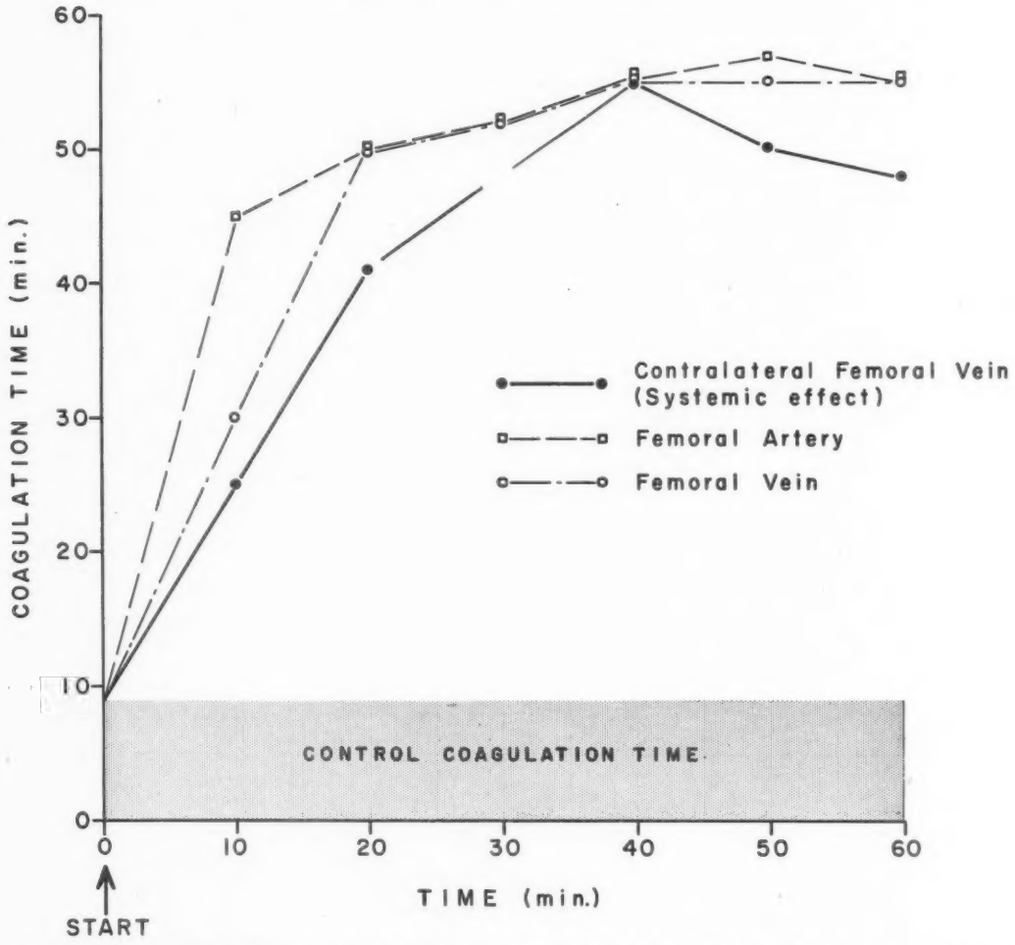


FIG. 5. Continuous injection of heparin (4 mg./100 cc. saline) into flowing iliac artery at 1.8 cc./min. (five dogs).

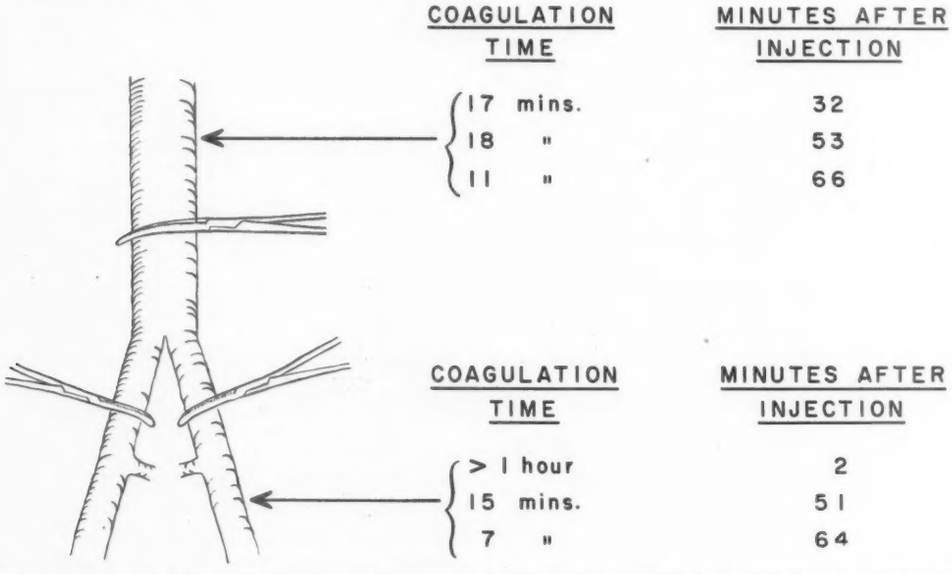


FIG. 6. Typical effect of injection of heparin (10 mg./100 cc. saline) into aorta prior to application of clamp. Samples for coagulation times were drawn at sites indicated at various times after injection and application of clamp. (Control = 9 minutes.)

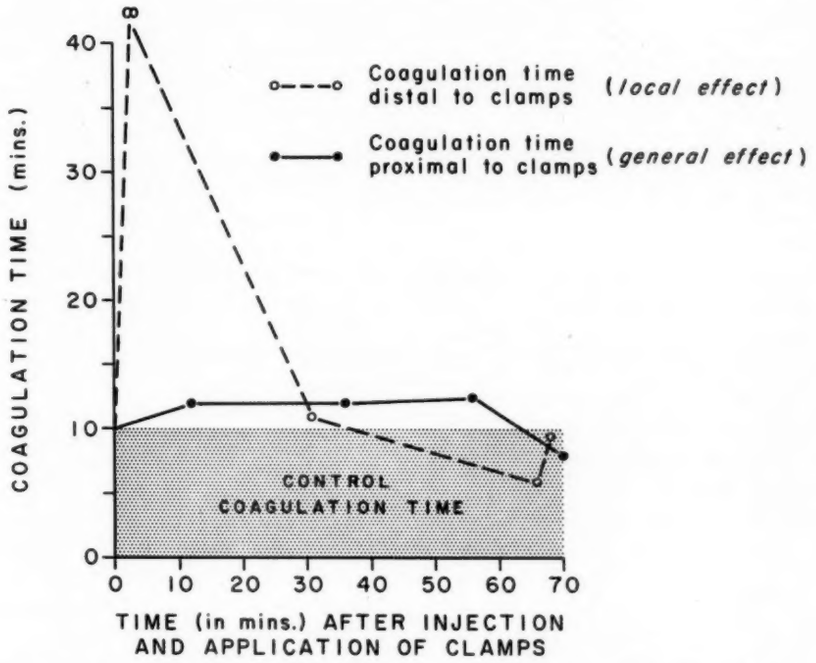

Coagulation time distal to clamps (local effect)
Coagulation time proximal to clamps (general effect)
COAGULATION TIME (mins.)
CONTROL COAGULATION TIME
TIME (in mins.) AFTER INJECTION AND APPLICATION OF CLAMPS

FIG. 7. Mean coagulation times after intra-aortic injection of heparin (10 mg./10 cc. saline) in twelve patients.

The use of a constant heparin perfusion in a proximally occluded vessel approximated, albeit temporarily, a relative local effect. Here it was definitely suggested that if a very minimal anticoagulant response can be obtained locally, perhaps the generalized response may not be obtained. However, the hairline difference in volume/minute of heparin delivered to produce a local or generalized response is so fine as to preclude its use. Finally, the anticoagulant response to this drug, to be effective, should be well above minimal response.

In the series of clinical cases studied the method of intra-arterial heparinization utilized suggests that repeated injections of heparin are necessary in the presence of prolonged arterial occlusion.

SUMMARY

The use of intra-arterial heparin by various methods experimentally is described. Each of these methods has been used in clinical vascular surgery.

"Regional heparinization" could not be obtained.

It is suggested that intra-arterial heparin, as used in arterial surgery, must be expected to produce a systemic response. The dangers inherent in its use as such must be considered.

REFERENCE

1. WYLIE, E. J., GARDNER, R. E., JOHANSEN, R. and MCCORKLE, H. J. An experimental study of regional heparinization. *Surgery*, 28: 29, 1950.

Ruptured Aneurysm of the Splenic Artery

CHARLES L. JAMES, M.D., *Birmingham, Alabama*

From the Surgical Service, Simon-Williamson Clinic, Birmingham, Alabama.

ANEURYSM of the splenic artery is an uncommon lesion and successful operation for its rupture has been rare. Therefore, this case is considered to be of sufficient interest to report.

CASE REPORT

R. G. (Hosp. No. A-4345), a sixty-two year old white woman, the mother of eleven children, was admitted to Highland Avenue Baptist Hospital on the night of October 23, 1955, with severe abdominal pain of about two hours' duration.

She had been treated for moderate hypertension for several years, the blood pressure being maintained at about 165/110. Mild edema of the feet, aching in the legs and slight soreness in the abdomen had been present over the preceding four years. Roentgenological studies of the stomach and gallbladder had been normal. For the past eight months she had had no complaints.

The present illness started by a sudden severe pain in the upper abdomen which persisted. She was hospitalized about two hours after the onset of symptoms. There was no nausea, vomiting or diarrhea.

On admission the temperature was 36°c., pulse 64, and blood pressure 138 systolic, 76 diastolic. The patient was somewhat obese and in moderate pain. The pain had become more generalized and now radiated into the back and left shoulder. The abdomen was without rigidity and moderate tympany was elicited on percussion. Peristaltic sounds were normal. Pelvic and rectal examinations were essentially negative. Femoral pulsations were normal bilaterally. There was no dependent edema.

Laboratory studies revealed a hemoglobin of 11 gm. per cent; erythrocytes, 3,950,000; leukocytes 10,400. The blood serologic test for syphilis was later reported negative. The urine contained a faint trace of albumin, no sugar, and an occasional hyaline cast microscopically.

During the night the patient continued to have abdominal pain partially relieved by narcotics. The following morning a roentgenogram of the abdomen showed a moderate amount of gas throughout the colon. No evidence of soft tissue density or calcification was seen. Other examinations including temperature, roentgenogram of the chest, electrocardiogram, serum amylase and blood sugar were normal. The leukocyte count had risen to 36,050 with 95 per cent neutrophils.

As the patient was expelling an enema given in preparation for a colon study, she went into collapse. The pulse became weak and rapid, she became pale and clammy, and the blood pressure was unobtainable. Vasopressors and a dextran infusion were given, followed by saline solution and a blood transfusion. As her condition improved somewhat, there was pain over the entire chest and abdomen, a little in the arms, but none in the legs.

The abdomen became moderately tense; peristaltic sounds persisted. Her condition appeared critical. In spite of another rapid transfusion the blood pressure did not rise above 80 mg. Hg systolic, and the pulse remained at about 120 per minute. The hemoglobin had dropped to 8 gm. per cent after the transfusion and her condition was deteriorating steadily. It became apparent that a major intra-abdominal hemorrhage was occurring, and immediate laparotomy was advised.

Under general anesthesia, with several simultaneous transfusions, the abdomen was opened through a long midline incision. Blood gushed forth in great quantities and profound shock immediately occurred. Spontaneous breathing ceased, blood pressure and peripheral pulse were unobtainable, but aortic pulsations were present. In spite of vigorous aspiration and sponging, the abdominal cavity could not be cleansed of blood sufficiently to determine the location of the bleeding. Some extravasated blood was present within the omentum and in the subserosa of the transverse mesocolon. Palpation along the aorta revealed no evidence of aneurysm. The brightest blood appeared from the left upper quadrant of the abdomen, and compression of the splenic pedicle slowed but failed to stop the hemorrhage.

Continued pressure of the splenic pedicle and compression of the abdominal aorta below the diaphragm controlled the hemorrhage, while continuous rapid transfusions were given through

multiple portals, until a weak peripheral pulse and blood pressure appeared. The abdominal incision was enlarged and the blood clots removed.

Blood had dissected extensively into the mesentery of the transverse colon, the splenic pedicle, the base of the omentum and the gastrocolic ligament. There were blood clots in the lesser peritoneal cavity and hemorrhagic dissection extended into the retroperitoneal tissues of the upper abdomen. The aorta was released and blood was seen to spurt from a ruptured aneurysm in the mid-portion of the splenic artery. The aneurysm was thin-walled, sacular and appeared to be about 2.5 cm. in diameter. The splenic vessels were ligated proximally; the spleen and aneurysm were removed.

Postoperatively two additional blood transfusions were required. The total volume of blood received by the patient during eighteen hours was 9,400 ml.

There was oliguria for eleven days, then diuresis developed. The blood urea nitrogen was 157 mg. per cent on the seventh day. Mild tarry diarrhea was present for a few days. The wound healed *per primam.*

She was unable to void spontaneously for several weeks. Psychic depression and apathy were severe, and difficulty in using the legs prevented walking for several weeks. She was discharged from the hospital on the twenty-fourth postoperative day. Thereafter improvement was gradual, and after one year she appeared normal except for hypertension.

REVIEW OF LITERATURE

This case, although not diagnosed preoperatively, presents many classic features of ruptured splenic artery aneurysm. It is the tenth reported case of this rare and usually fatal condition with surgical recovery.

The first report of an aneurysm of the splenic artery was by Beaussier [*1*] in 1770. It was discovered in a female cadaver. Christ [*2*] reported the second case in 1847. The first successful operation for splenic artery aneurysm was performed by Winkler [*3*] in 1903 on a twenty-five year old woman who had abdominal pain, anemia and an enlarged spleen. Multiple unruptured aneurysms the size of "pigeon eggs" were found in the splenic vessels. Splenectomy resulted in recovery.

Guy [*4*] states that Höegler in 1920 first clinically diagnosed the condition on the basis of left upper quadrant abdominal pain associated with a palpable mass, a systolic bruit and roentgenologically a pulsating filling defect on the lesser curvature of the stomach. The diagnosis was confirmed at autopsy.

In 1922 Marshal [*5*] diagnosed pancreatic cyst in a young woman with a previous gunshot wound in the upper abdomen. At operation he opened the mass and encountered fierce bleeding. He packed the wound and eight days later reoperated for recurrent hemorrhage. The mass proved to be an aneurysm of the splenic artery. The artery was ligated at each end of the aneurysm and the patient recovered.

Faehrmann [*6*] in 1924 performed exploration on a man for splenomegaly associated with left upper quadrant abdominal pain and anemia. Free blood was present in the peritoneum. On manipulation of the very large spleen, tremendous bleeding occurred and splenectomy was quickly performed. Old blood clots were noted in the splenic bed. Three days later the patient died, and necropsy revealed a massive hemorrhage from a large ruptured splenic arterial aneurysm proximal to the site of vessel ligation.

In 1931 Mallet-Guy [*7*] reported the first case of ruptured splenic artery aneurysm diagnosed preoperatively and confirmed at operation, but the patient died.

Surgical recovery after rupture was first reported by Brockman [*8*] in 1930, in a sixty-nine year old woman. The rupture had occurred into the lesser omental cavity. Splenectomy with ligation of the vessels proximal to the aneurysm resulted in recovery.

Parsons [*9*] reported the next successful operation in 1937. He removed the aneurysm and the spleen from a man, with recovery. A fatal recurrent hemorrhage occurred from another aneurysm two years later in the proximal portion of the splenic artery.

MacLeod and Maurice [*10*] in 1940 first reported surgical recovery in a pregnant woman with spontaneous rupture of the splenic artery. The massive hemorrhage made examination difficult, but no aneurysm was identified. Gillam [*11*] in 1942 reported the first surgical recovery in a pregnant woman with rupture of a definitely identified aneurysm of the splenic artery. The baby was stillborn.

Dobson et al. [*12*] in 1947 reported the next recovery after operation, and in 1952 Milone [*13*] and Tagart [*14*] each reported one additional case. Milone's case is of interest because the patient underwent several surgical procedures before the cause of the hemorrhage was found. Wood and Hull [*15*] in 1954, Downs et al. [*16*] in 1955 and Parrella [*17*] in 1955 each reported a case, which together with the one reported herein make ten with operative recov-

ery reported in the literature, excluding MacLeod and Maurice's case. When not complicated by rupture, surgery has more often been successful. Colangelo [*18*] in 1955 found thirty cases reported since Winkler's first successful operation for non-ruptured splenic artery aneurysm.

INCIDENCE

The incidence of aneurysm of the splenic artery was reported as 0.08 per cent in 32,768 autopsies by Schroeder [*19*], 0.05 per cent in 41,437 autopsies by Lindboe [*20*], and .02 per cent in 33,810 autopsies by Sperling [*21*]. In 1953 Owens and Coffey [*22*], in a comprehensive review of the literature, collected 198 cases of splenic artery aneurysms and added six cases, bringing the total to 204 cases. In 1955 Colangelo collected twelve additional cases, including one of his own, to bring the total to 216 cases.

The splenic artery is one of the more common branches of the abdominal aorta to be affected by aneurysms. Palmer [*23*] in 1950 collected 150 cases of aneurysm of the splenic artery, seventy-seven of the renal artery and sixty-nine of the hepatic vessels. In a review of 33,810 autopsies Sperling found 393 aneurysms, seventy of which were in the abdomen. Forty-eight involved the abdominal aorta (twenty-six ruptured). Of the remaining twenty-two, nine were of the iliac, seven of the splenic, two each of the celiac and renal, and one each of the hepatic and superior mesenteric arteries. The average age at diagnosis is about forty-eight years, and about 45 per cent of recorded cases have been ruptured [*22*]. The lesion in the splenic artery occurs about twice as frequently in females as in males, while when located elsewhere in the body the ratio of males to females is about 5:1.

A relationship between pregnancy and rupture is suggested by the observation that about 24 per cent of the female patients who died following rupture of this aneurysm were pregnant, usually at or near delivery [*22*]. Prior to 1955 Poidevin [*24*] found about thirty cases of rupture reported in pregnant women, with only two survivors.

PATHOLOGY

Aneurysms of the splenic artery vary in size from a few millimeters to many centimeters in diameter. Generally they are small. They tend to be along the main trunk of the vessel, but occur occasionally at the bifurcation or in the hilar branches. Sherlock and Learmonth [*25*] reported them to be single in 74 per cent of cases, and multiple in 26 per cent.

After hemorrhage the gross findings will vary with the direction in which dissection occurs, and its rapidity. If leakage has occurred previously, there may be dense adhesions between the aneurysm and stomach, transverse colon, spleen and pancreas. The blood tends to dissect into the retroperitoneal tissues, along the gastrocolic ligament, the mesocolon, into the splenic pedicle and into the lesser omental bursa. Tamponade may occur temporarily followed by massive hemorrhage that escapes through the foramen of Winslow or directly into the greater peritoneal cavity through the gastrocolic ligament. Erosion may occur into the adherent stomach or colon, or even into the splenic vein.

Microscopically, the wall of the diseased vessel usually shows degenerative changes, greatest in the media where there may be focal or generalized necrosis, fibrosis or even calcification as in advanced arteriosclerosis. The elastic laminas may be deficient. The media may be thin, and thickening of the intima may occur. Atheromatous or arteriosclerotic changes may be absent, especially in younger persons. Possible congenital defects in the vessel wall are difficult to demonstrate pathologically.

ETIOLOGY

The etiology is usually obscure. Congenital defects in the arterial wall, arteriosclerotic changes, trauma, infection, embolic phenomena and portal hypertension have been considered as causative factors. The occurrence of multiple small aneurysms, especially at the bifurcation of the splenic artery, suggests the possibility of a congenital defect such as occurs in the circle of Willis, particularly in younger persons.

Like aneurysms elsewhere in the abdomen, the great majority are on an arteriosclerotic basis, particularly in the main trunk of the splenic artery.

A history of direct trauma is rare. In two reported cases [*5,22*] the aneurysm followed gunshot wounds of the upper abdomen. Syphilis is rarely the etiology. Inflammation or arteritis may be causes. Infarctions resulting from emboli may produce aneurysms, particularly those in the hilar branches.

Splenomegaly is frequently observed in these patients and persistent hypertension in the splenic artery secondary to it or coincident with portal venous hypertension is a possible causative factor. Less prolonged increase in the blood pressure occurs during delivery and physical exertion.

Remizov [*26*] pointed out that rupture occurs often when there is temporary or sustained increase in intra-arterial blood pressure in the presence of a weakened vessel wall.

SYMPTOMS

Before rupture there may be no symptoms or only vague crampy left upper abdominal pain. Symptoms may be related to adjacent organs and simulate disorders of the stomach, duodenum or gallbladder. Occasionally gastrointestinal bleeding has been reported.

After rupture, the symptoms become acute and often catastrophic. Brockman states the rupture may be in two phases: first, leakage from the aneurysm locally in the region of the splenic pedicle or lesser omental sac. Clotting or tamponade may temporarily halt the hemorrhage, or adhesions may even form. Hours or weeks later the second phase occurs: then dissection proceeds rapidly. The abdominal cavity fills with blood or the bleeding may occur into an adjacent hollow viscus, usually the stomach or colon. Owens and Coffey, in their ninety-four collected cases, found fifty-one with single rupture and forty-three with two-stage rupture.

In the first phase there is sudden, often crampy abdominal pain in the upper left quadrant of the abdomen. Nausea and vomiting may occur. As dissection proceeds, the area of pain widens and it may radiate into the tip of the left shoulder or into both shoulders. After several hours, if the hemorrhage is contained, symptoms may almost disappear. Immediately, if the hemorrhage is not contained (or if contained, then with the second phase), the massive hemorrhage proceeds and the classic picture of hemorrhagic shock develops, often with a rapid fatal outcome.

DIAGNOSIS

A correct preoperative diagnosis has rarely been made because the history and physical findings usually are not suggestive. Before rupture the condition simulates psychosomatic, biliary or gastrointestinal disorders. After rupture it must be differentiated from other vascular accidents such as mesenteric thrombosis, embolus to the lung or other viscus, myocardial infarction, ruptured aortic aneurysm; from ruptured or perforated viscus such as the spleen, duodenum, pregnant fallopian tube or gravid uterus, and from other conditions such as acute pancreatitis, volvulus, separation of the placenta and acute intestinal obstruction. In the infrequent incidence of rupture into the stomach or colon the diagnosis may be confused with ruptured esophageal varix or bleeding duodenal ulcer. Rupture of an aneurysm complicating pregnancy has usually been confused with either rupture of the uterus or premature separation of the placenta.

Occasionally the aneurysm is palpable. It may be expansile and the presence of a bruit may aid in the diagnosis. Roentgenographic demonstration of calcification in the aneurysm appears as a calcific ring surrounding scattered mottled densities. By laminagraphy a stalk-like appearance to the calcific circle characterizes aneurysm. Near the splenic hilum the calcified aneurysm may show respiratory movement fluoroscopically. If large, external indentation of the posterior gastric wall may be demonstrated with barium study, and this sign is of greater value if the mass pulsates. Urograms occasionally are helpful and more recently the aneurysm has been localized with aortographic studies.

TREATMENT

Unruptured aneurysms should be treated surgically since about 45 per cent of them rupture. Surgical treatment is usually not difficult and consists of excision of the aneurysm with splenectomy. Splenectomy without excision of the aneurysm, ligation of the aneurysm with or without its removal and without splenectomy have all been performed, but have no advantages and are not recommended when removal of the spleen and aneurysm are feasible. The surgical mortality rate in the past has been high but is now almost nil.

After rupture the treatment is hazardous. Ligation of the splenic vessels proximal to the aneurysm and close to the celiac axis with removal of the aneurysm and spleen is the treatment of choice and has been employed in most of the successful cases. Ligation of the splenic

artery at each end of the aneurysm has been successful. Simple packing of the ruptured aneurysm has been uniformly fatal. When complicated by advanced pregnancy, it may be necessary to evacuate the uterus to obtain access to the bleeding aneurysm. The fetus has consistently been stillborn when the aneurysm is ruptured.

After rupture the outcome has been almost uniformly fatal, but recent technical advances have reduced the surgical mortality rate.

COMMENTS

The case reported herein presented several typical features. The patient was a female with hypertension and some arteriosclerotic changes in the larger vessels. The rupture was in two phases, the final one occurring during physical exertion. The clinical picture after rupture was classic.

Had the diagnosis been suspected earlier, the operation could have been performed after the first phase of rupture with much less risk, since the quiescent stage that often occurs between the two stages of rupture is the time of choice for surgery.

At operation, if the diagnosis is not previously suspected, the source of the massive hemorrhage is often difficult to locate. Blood in the transverse mesocolon or blood that escapes through the foramen of Winslow suggests splenic artery aneurysm.

These aneurysms, like degenerative diseases elsewhere in the vascular system, will probably be encountered with increasing frequency as the population grows older.

SUMMARY

1. Aneurysm of the splenic artery is not common. When rupture occurs it is usually fatal unless aggressive surgical treatment is promptly initiated.

2. The diagnosis before rupture is very difficult unless the lesion is kept in mind and a pulsating mass is palpable or the aneurysm is demonstrable roentgenologically. Rupture may be suspected if there is initial sudden severe pain in the left upper quadrant with mild shock followed by a temporary period of improvement and then sudden and almost complete collapse with findings characteristic of massive intra-abdominal hemorrhage.

3. The treatment of choice is massive transfusion and supportive measures with prompt removal of the ruptured aneurysm and spleen.

4. After rupture only nine cases have been reported with recovery; a tenth case is added herein.

Acknowledgment: Grateful acknowledgment is made to Dr. Gordon L. Ross, internist, Drs. L. W. Funderburg and William O. Romine, anesthesiologists, and Dr. Steriling W. Edward, III, Assistant Professor of Surgery at the University of Alabama Medical Center, for their able assistance.

REFERENCES

1. Beaussier, M. Quoted by Owens, J. C. and Coffey, R. J. [22].
2. Christ, E. Quoted by Owens, J. C. and Coffey, R. J. [22].
3. Winkler, V. Ein Fall von Milzexstirpation wagen Aneurysma der Arteria lienalis. *Zentralbl. Chir.*, 1: 257, 1905.
4. Guy, C. C. Aneurysm of the splenic artery. *Surgery*, 5: 602, 1939.
5. Marshall, C. J. Traumatic aneurysm of the splenic artery, rupture, ligature. *Brit. J. Surg.*, 9: 570, 1922.
6. Faehrmann, I. and Ugrümow, B. Ein fall von aneurysma der arteria lienalis. *Arch. klin. Chir.*, 137: 193, 1925.
7. Mallet-Guy, P. Quoted by Owens, J. C. and Coffey, R. J. [22].
8. Brockman, R. St. L. Aneurysm of the splenic artery. *Brit. J. Surg.*, 17: 692, 1930.
9. Parsons, C. G. A case of ruptured aneurysm of the splenic artery with recurrence. *Brit. J. Surg.*, 24: 708, 1937.
10. MacLeod, D. and Maurice, T. Rupture of a branch of the splenic artery associated with pregnancy. *Lancet*, 31: 924, 1940.
11. Gillam, J. F. E. Ruptured aneurysm of the splenic artery during pregnancy. *Brit. M. J.*, 1: 69, 1942.
12. Dobson, L., Morse, D. and Covert, J. Aneurysm of the splenic artery. *Stanford M. Bull.*, 5: 39, 1947.
13. Milone, S. L'aneurisma dell'arteria splenica. *Arch. ital. chir.*, 75: 427, 1952.
14. Tagart, R. E. B. Ruptured splenic arterial aneurysm. *Brit. J. Surg.*, 39: 469, 1952.
15. Wood, W. S. and Hull, B. Rupture of spleen in subacute bacterial endocarditis. *Arch. Int. Med.*, 93: 633, 1954.
16. Downs, C. R., Hopeman, A. R., Todd, W. A. Jr. and Mathewson, C. M., Jr. Aneurysm of splenic artery. *Ann. Surg.*, 141: 268, 1955.
17. Parrella, G. S. Rupture of splenic artery aneurysm. *New England J. Med.*, 252: 814, 1955.
18. Colangelo, C. Aneurysm of the splenic artery. *Illinois M. J.*, 108: 223, 1955.
19. Schroeder, C. Quoted by Sperling, L. [21].
20. Lindboe, E. F. Aneurysm of the splenic artery

diagnosed by x-ray and operated upon with success. *Acta chir. scandinav.*, 72: 108, 1932.

21. SPERLING, L. Aneurysm of the splenic artery. *Surgery*, 8: 633, 1940.
22. OWENS, J. C. and COFFEY, R. J. Aneurysm of splenic artery, including a report of six additional cases. *Surg., Gynec. & Obst.*, 97: 313, 1953.
23. PALMER, T. H. Aneurysm of splenic artery. *New England J. Med.*, 243: 989, 1950.
24. POIDEVIN, L. O. S. Rupture of splenic artery aneurysm in pregnancy. *M. J. Australia*, 1: 922, 1955.
25. SHERLOCK, S. P. V. and LEARMONTH, J. R. Aneurysm of the splenic artery with an account of an example complicating Gaucher's disease. *Brit. J. Surg.*, 30: 151, 1942.
26. REMIZOV, A. A. Quoted by Owens, J. C. and Coffey, R. J. [22].

"A human body was publicly dissected for the first time in many hundreds of years in Venice in 1308. At first there was an outcry against the sacrilegious immorality of such a proceeding. In the course of years, however, the practice became accepted and remarkably elaborated. Two ecclesiastical dignitaries and two distinguished surgeons would select carefully a young and healthy criminal. Early on a fine morning he was brought by his warders, who were very proud of him, to the hall of the university. There was gathered a great concourse of people. All the dignitaries of Church and State were assembled, with professors of the university and physicians and surgeons in their long robes. Great merchants and their wives were there with their pretty daughters, the merchants very conscious of the fact that they had been singled out for invitation to a most important social function, their wives just as conscious of the fact that their pretty daughters were ogling apprentices and students who needed no such encouragement. There was a buzz of conversation and all eyes turned to the high platform to which the condemned criminal had been led. The prolonged rites proper to the occasion were begun. They ended with the certain promise of spiritual indulgence as some compensation to the soul of the prisoner, whose bodily frame was to be subjected to no little indignity. The ecclesiastics stepped back and gave place to the executioner. The young man was efficiently strangled, and thus transferred from the protection of the Church to the care of the waiting stewards. His body was extended, face upwards, upon a low table. The chief surgeon seated himself upon a chair raised high above the body. A grey-bearded colleague on his right began to read from a vellum roll. He described first the four abdominal muscles. The steward's knife opened widely the abdominal flesh, and the long ivory wand, which the chief surgeon held, waved vaguely. There was a gratifying murmur of awed interest from the intent audience, a murmur which reached its height a little later as the wand waved even more vaguely about a multi-lobed liver and its double bile-duct. So it went on for the whole day, with frequent pauses for rest and for food and drink." (From GRAHAM, H. Surgeons All, p. 156. New York, 1957. Philosophical Library, Inc.)

The Status of Head and Neck Surgery in Hospitals Approved for Four-Year Residency Training

ARTHUR G. JAMES, M.D.,* *Columbus, Ohio*

THE Society of Head and Neck Surgeons is an organization whose main function is the furtherance of surgery of the head and neck areas.

A questionnaire was prepared by this Society and sent to the chiefs of the surgical services in 213 hospitals in the United States which had four-year approved surgical residencies. Seventy of these institutions were also approved for ear, nose and throat residency programs. The list of hospitals was obtained from the *Journal of the American Medical Association*, September 22, 1956. One hundred four of these were hospitals in university centers and 109 were not affiliated with medical schools.

The following is a copy of the questionnaire that was submitted:

QUESTIONNAIRE
ON POSTGRADUATE TRAINING IN HEAD AND NECK SURGERY

I. In which department of your institution are the following surgical procedures performed?
   1. Radical neck dissection
   2. Partial laryngectomy (laryngofissure)
   3. Total laryngectomy
   4. Glossectomy
   5. Excision of cancer of the lip
   6. Parotidectomy
   7. Thyroidectomy
   8. Resection of the maxilla for cancer
   9. Resection of the mandible for cancer
   10. Intraoral surgery for cancer
   11. Excision of thyroglossal and branchiogenic cysts

II. In your institution is any effective training in head and neck surgery provided for ear, nose and throat residents?

   If so, by whom?

III. What training do you think a surgeon should have, to be qualified to perform the head and neck surgical procedures listed above?

Name.....
Official Title.....
Institution.....

MATERIAL

The answers to question I were grouped into six categories: general surgery; ear, nose and throat; ear, nose and throat and/or general surgery; plastic surgery; plastic and/or general surgery; and varied combinations of services.

Many answers indicated that some of the procedures were performed on two services, or by cooperation between departments, i.e., ear, nose and throat plus general surgery, or plastic plus general surgery. There were also a number of institutions in which several services combined efforts in treating problems of the head and neck and these were classified simply as varied combinations of services.

Radical neck dissection:

General surgery	113
Ear, nose and throat	0
Ear, nose and throat and/or general surgery	16
Plastic surgery	4
Plastic and/or general surgery	20
Varied combinations of services	15

* Chairman, Postgraduate Committee, Society of Head and Neck Surgeons. Members of the Committee are: Eugene W. Friedman, M.D., Mason Morfit, M.D., Samuel L. Perzik, M.D., Kenneth L. Pickrell, M.D. and Danely P. Slaughter, M.D.

Partial laryngectomy:
- General surgery 17
- Ear, nose and throat 121
- Ear, nose and throat and/or general surgery 20
- Plastic surgery 0
- Plastic and/or general surgery 6
- Varied combinations of services 11

Total laryngectomy:
- General surgery 20
- Ear, nose and throat 95
- Ear, nose and throat and/or general surgery 33
- Plastic surgery 1
- Plastic and/or general surgery 0
- Varied combinations of services 19

Glossectomy:
- General surgery 115
- Ear, nose and throat 9
- Ear, nose and throat and/or general surgery 6
- Plastic surgery 11
- Plastic and/or general surgery 16
- Varied combinations of services 11

Excision of cancer of the lip:
- General surgery 114
- Ear, nose and throat 0
- Ear, nose and throat and/or general surgery 3
- Plastic surgery 10
- Plastic and/or general surgery 29
- Varied combinations of services 12

Parotidectomy:
- General surgery 123
- Ear, nose and throat 1
- Ear, nose and throat and/or general surgery 11
- Plastic surgery 8
- Plastic and/or general surgery 16
- Varied combinations of services 9

Thyroidectomy:
- General surgery 160
- Ear, nose and throat 0
- Ear, nose and throat and/or general surgery 3
- Plastic surgery 1
- Plastic and/or general surgery 1
- Varied combinations of services 3

Resection of maxilla for cancer:
- General surgery 89
- Ear, nose and throat 5
- Ear, nose and throat and/or general surgery 14
- Plastic surgery 20
- Plastic and/or general surgery 11
- Varied combinations of services 29

Resection of the mandible for cancer:
- General surgery 118
- Ear, nose and throat 3
- Ear, nose and throat and/or general surgery 5
- Plastic surgery 20
- Plastic and/or general surgery 18
- Varied combinations of services 22

Resection for Intraoral Cancer:
- General surgery 94
- Ear, nose and throat 7
- Ear, nose and throat and/or general surgery 9
- Plastic surgery 16
- Plastic and/or general surgery 15
- Varied combinations of services 27

Excision of thyroglossal and branchiogenic cysts:
- General surgery 136
- Ear, nose and throat 1
- Ear, nose and throat and/or general surgery 16
- Plastic surgery 1
- Plastic and/or general surgery 7
- Varied combinations of services 7

The answers to question 11 regarding the effective training for ear, nose and throat residents in head and neck surgery were sometimes difficult to evaluate. In many instances the institution did not have an approved ear, nose and throat residency. When training was offered, an attempt was made to evaluate which service or services supplied this teaching. The final summary indicated that in 123 hospitals there was no effective training. Training of varying degrees was given in thirty-seven hospitals. In sixteen instances, this was supervised by the ear, nose and throat departments, in six by the general surgical services, and in fifteen institutions the training represented a joint effort of several services. It is apparent from these figures that for the main, the ear, nose and throat residents are receiving meager training in this field of surgery.

Most of the answers to question 111 regarding postgraduate training indicated that a general

surgical background was necessary for the best performance in surgery of the head and neck. The following categories of answers were received: board qualification in general surgery, seventy-one; general surgery plus specialized head and neck training, forty; general surgery plus ear, nose and throat training, thirteen; general surgery plus plastic training, eleven; and general surgery plus varied combinations of specialized training, thirty-one.

One hundred seventy-six of the 213 hospitals polled responded, for a percentage of 83 per cent answers. One hundred four of the 213 hospitals were affiliated with university centers. Eighty-nine or 85 per cent of these answered. One hundred nine of the 213 hospitals were not in university centers. Eighty-seven or 80 per cent of these answered. The results therefore represent the opinions of approximately 83 per cent of the chiefs of the approved four-year surgical residencies in the United States.

CONCLUSIONS

1. Most of the head and neck surgery in the 176 hospitals that participated in the poll was performed on the general surgical services.
2. In these institutions very little effective, organized training in head and neck surgery was offered the ear, nose and throat resident.
3. Most surgical chiefs who answered thought that a general surgical background was essential in the postgraduate training of the surgeon who intends to perform adequate head and neck surgery. Forty per cent in fact stated that full general surgical board accreditation should be required.

"Another celebrated Italian anatomist, Andrea Cesalpino (1519 or 1524–1603) was professor of medicine at Pisa and physician to Pope Clement VIII. He is a most important historic personality because a long standing bitter controversy has existed as to whether Cesalpino or Harvey was the true discoverer of the circulation. The Italian historians, particularly, are vehement in their contention that the priority of this important discovery belongs to Cesalpino. In a recent monograph on this subject Arcieri made the following impressive statement: 'Unfortunately there is an unlimited number of historians, ever renowned, who elect themselves critics in the question, obviously without having performed the hard task of reading all the Cesalpinian works.' While some historians acknowledge that Cesalpino conceived the general idea of the circulation of the blood they contend that he presented no experimental evidence to this effect.

"However, Arcieri denied this allegation and quoted directly and extensively from Cesalpino's works to the effect that his conclusions were based on detailed dissections, observations on the motions of the heart and vessels and on clear reasoning. Arcieri's monograph merits careful study. Corwin in a recent publication supported the Italian contention of Cesalpino's priority. . . Cesalpino's observations certainly indicate that he was possessed of a remarkably clear idea of the general scheme of the circulation." (From: WILLIUS, F. A. and DRY, T. J. A History of the Heart and the Circulation. Philadelphia, 1948. W. B. Saunders Co.)

Management of Goiter in Children

CARL P. SCHLICKE, M.D. AND ROBERT BERGHAN, M.D., *Spokane, Washington*

From the Department of Surgery and the Department of Internal Medicine of the Rockwood Clinic, Spokane, Washington.

THE management of goiter in children presents a problem which differs in many respects from the conventional approach to goiter in adults. Since goiter in childhood is not common, few doctors acquire extensive personal experience in dealing with it, yet it is important for those upon whom this responsibility falls to recognize the differences which exist.

Congenital goiter may be symptomatic at birth but sometimes does not cause trouble until later in childhood. It not infrequently results in serious respiratory obstruction and in rare instances may be the site of a malignant tumor. [*11*]. A colloid goiter which might pass almost unnoticed in an adult may bring upon a sensitive child the ridicule of playmates with distressing psychologic repercussions. The associated hypothyroidism, which if uncorrected in an adult might only result in sluggishness and gain in weight, can interfere with the growth and maturation of a child. The hazard of carcinoma, well known to exist in nodular goiter, is even greater in the child than in the adult [*15,17*].

The hyperthyroid state, for which a variety of therapeutic modalities is now available, presents a particularly complex dilemma. The disease is often severe and if inadequately controlled may retard growth and development. Surgery in children is associated with a higher rate of morbidity, mortality and recurrence than in adults [*9*]. Treatment with antithyroid drugs is of necessity prolonged, requires constant supervision and its results are unpredictable. The use of radioactive iodine in children is generally condemned [*10*], but x-ray therapy occasionally is advised when other conservative measures fail [*13*].

Malignant tumors of the thyroid often pass unrecognized while the unfortunate young host has tonsils and adenoids removed and is given all the latest antibiotics for "swollen glands." When the true nature of the condition is appreciated general agreement regarding details of treatment is lacking. There is a widespread tendency to regard the tumors as relatively benign, yet 20 per cent of the children in whom they occur are dead within five years [*19*]. In all types of goiters in children the problem is more serious than in adults [*15*]. With these considerations in mind we determined to assemble such information as was available to us concerning the treatment of goitrous children in this community.

TABLE I
CLASSIFICATION OF CASES

Type of Goiter	No. of Patients		
	Girls	Boys	Total
Congenital	1	0	1
Colloid	2	0	2
Adenomatous	3	1	4
Toxic	5	2	7
Chronic thyroiditis	3	2	5
Carcinoma	4	0	4
Total	18	5	23

MATERIAL

A review was made of the records of all patients under fifteen years of age admitted to the three principal Spokane hospitals for the treatment of goiter during the seven year period from 1950 through 1956. Twenty-three children comprise the basis for this report. There were eighteen girls and five boys. The youngest child treated was five years old. The youngest child operated upon was seven. The

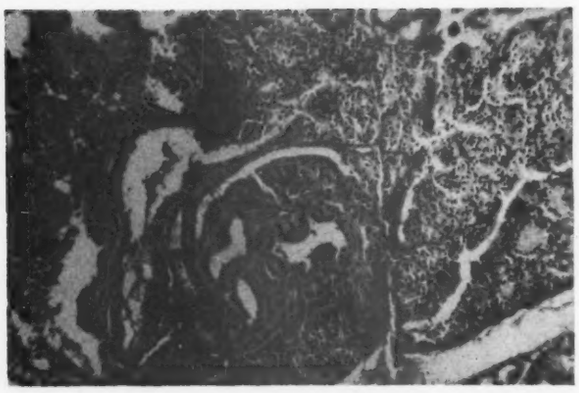


FIG. 1. Photomicrograph of tissue removed from patient with congenital goiter.

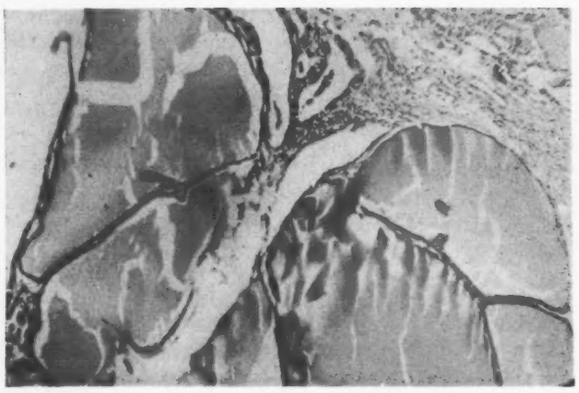


FIG. 2. Photomicrograph of tissue removed from patient with colloid goiter.

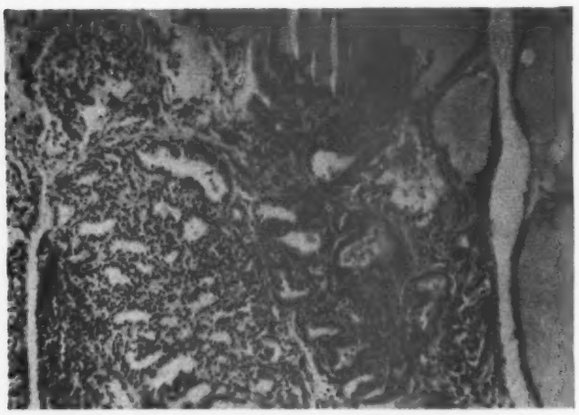


FIG. 3. Photomicrograph of tissue removed from child with adenomatous goiter.

FIG. 4. Photomicrograph of tissue removed from patient with diffuse goiter with hyperthyroidism.

remainder ranged in age between nine and fourteen years. Diagnoses included: congenital goiter, one patient; colloid goiter, two patients; adenomatous goiter, four patients; toxic goiter, seven patients; chronic thyroiditis, five patients; and carcinoma, four patients. (Table 1.)

TREATMENT AND RESULTS

Congenital Goiter. The nine year old girl having this condition was subjected to subtotal thyroidectomy for cosmetic reasons, 75 gm. of tissue being removed. (Fig. 1.) Convalescence was uneventful and the child has been well since operation. Preoperative diagnosis was colloid goiter.

Colloid Goiter. There were two children exhibiting this condition. A nine year old boy with epilepsy and associated hypothyroidism was treated medically. A thirteen year old girl in whom goiter was regarded as adenomatous clinically underwent subtotal thyroidectomy with removal of 120 gm. of tissue. (Fig. 2.) Her convalescence was uneventful and she has been quite well since.

Adenomatous Goiter. There were four children in this group and all were subjected to operation. A girl, aged seven, had a solitary nodule removed. A boy, aged ten, underwent resection of the right lobe (Fig. 3) (the left being absent), and the remaining two girls, aged eleven and thirteen respectively, underwent subtotal thyroidectomy. Preoperative diagnoses were obvious, convalescence uneventful and all patients have done well since operation.

Toxic Goiter. The enlargement of the gland in five of these children was diffuse and in two it was nodular. Two boys, aged five and nine respectively, in whom the disease was so mild as to be almost questionable were treated medically. One girl, aged nine, with exophthalmic goiter, in whom thyrotoxicosis was severe (basal metabolic rate +38 to +100, cholesterol 70 mg. per cent, pulse 132) obtained a good

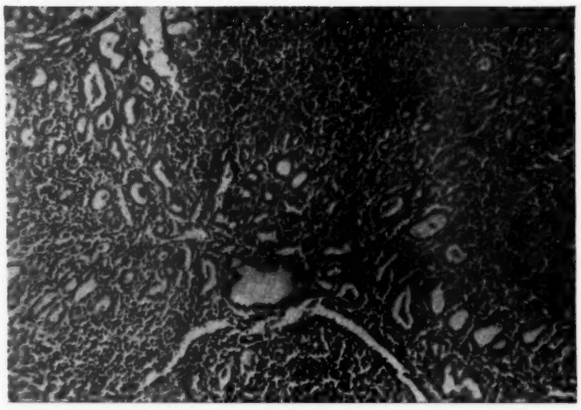


FIG. 5. Photomicrograph of tissue removed from patient with struma lymphomatosa.

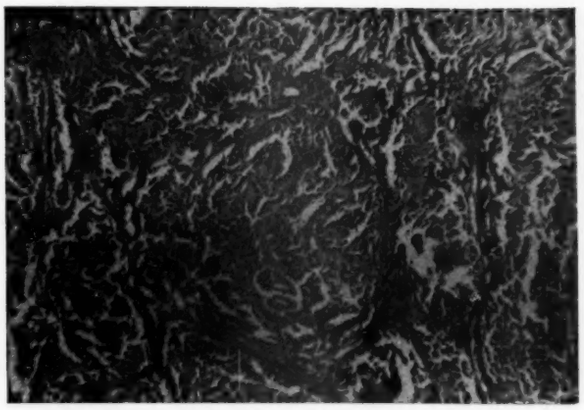


FIG. 6. Photomicrograph of tissue removed from patient with papillary adenocarcinoma of the thyroid.

immediate response to tapazole,® but after two months her family became impatient and took her to another physician who performed subtotal thyroidectomy. Convalescence was uneventful and the child has been well and has developed normally during the six years which have elapsed since operation. The remaining patients, three girls aged thirteen and one aged fourteen, were also treated surgically. These four girls had severe hyperthyroidism. Two were thought to exhibit retarded development. In all four, attempts at control (several months to two years) with antithyroid drugs had been ineffective. Although some improvement occurred, nothing approaching euthyroidism was achieved. All four girls received iodine in the immediate preoperative period. In all four cases the operation was a nerve-racking experience for the surgeon because of the friable, vascular character of the glands (Fig. 4) and the patients' response, which in one child was so alarming as to lead to discontinuance of the procedure after removal of the first lobe had been completed; the second lobe was removed uneventfully two months later. Convalescence in each case was stormy. The systolic blood pressure exceeded 170 mm. Hg during the first two postoperative days in every case, rising to over 200 mm. Hg in two patients, the pulse rate ranging between 170 and 210. All four patients received oxygen, antibiotics and sedation. Three were given ACTH which, in at least one patient, was regarded as life saving in the presence of a severe crisis. In one child atelectatic pneumonia developed on the second postoperative day and tracheal aspiration was required. Another child required tracheotomy on the fourth postoperative day because of increasingly labored respiration, deepening cyanosis and impending asphyxia, although two days later the vocal cords were found to be moving normally and the airway proved adequate when the tracheotomy tube was removed five days after its insertion. Happily there were no deaths in this small but hectic series and no instances of permanent cord paralysis or tetany; each of the patients ultimately obtained a gratifyingly satisfactory end result.

Thyroiditis. Patients in this category included two boys, aged ten and thirteen and three girls, aged nine, ten and thirteen. Preoperatively, carcinoma was suspected in four of these children because of the hard, nodular character of their glands. In one child the goiter was known to have been present for two months and in the other children for one or more years. In three children there were symptoms suggestive of mild hyperthyroidism (unconfirmed by laboratory studies) and in one child hypothyroidism was present and growth and maturation were retarded. Unilateral total lobectomy was performed on two patients, resection of isthmus on one and conservative subtotal thyroidectomy on two. Lymph node biopsy in three instances was also performed. In all these children frozen and fixed sections of the gland showed chronic thyroiditis (Hashimoto's type) (Fig. 5), and the lymph nodes which were removed showed benign hyperplasia. Convalescence was uneventful except for protracted bleeding from the skin edges in one patient. One patient received x-ray therapy postoperatively. Three have required administration of thyroid extract since operation; otherwise all have been well and have developed normally.

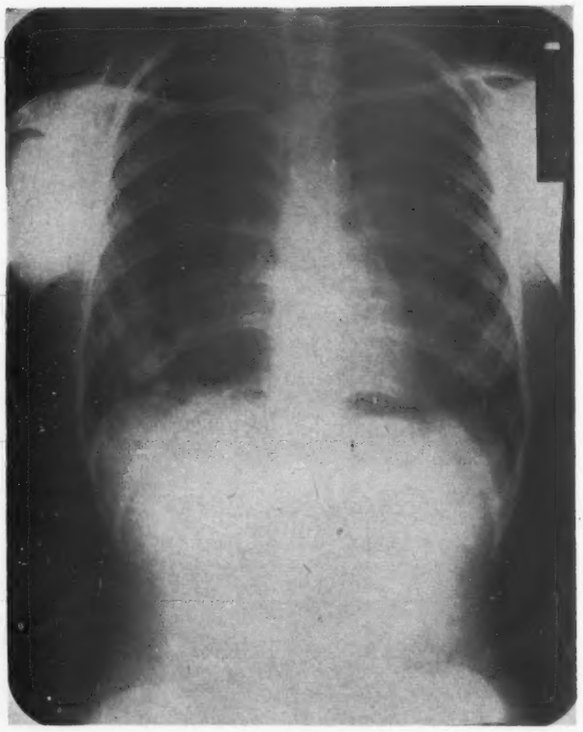


FIG. 7. Photograph of chest film of patient with pulmonary metastasis secondary to carcinoma of the thyroid.

Carcinoma. All of the patients who had malignant tumors of the thyroid gland were girls, two aged nine and two aged thirteen. On admission, the glands in two girls were thought to be adenomatous while in the other two the glands were suspected of being malignant because of the presence of enlarged regional lymph nodes. The following operations were carried out: unilateral total lobectomy; total right lobectomy, resection of the left lobe and biopsy of a lymph node; total thyroidectomy and simultaneous bilateral radical neck dissection; total thyroidectomy and subsequent bilateral neck dissection. Pathological study revealed papillary adenocarcinoma in all of the glands (Fig. 6), with metastasis to lymph nodes in both patients subjected to neck dissection, but no metastasis was noted in the patient who had only node biopsy. Three patients received x-ray therapy postoperatively and one of these was also given radioactive iodine. Convalescence was uneventful in all cases. Each of the patients who had undergone radical neck dissection had further cervical node biopsy the following year; both biopsies were reported as inflammatory hyperplasia without evidence of tumor. However, at this writing (four years later), one of these patients has multiple small pulmonary metastatic lesions. (Fig. 7.) The other three patients are living without evidence of recurrence one, five and six years, respectively, since their operations. One of them has mild, chronic hypoparathyroidism which is kept under satisfactory control by oral administration of calcium occasionally supplemented by dihydrotachysterol. The other two are quite well and one of them is now married and the mother of two children.

TABLE II
TREATMENT

Type of Treatment	No. of Patients	Type of Goiter
Medical	1	Colloid
	2	Toxic
Surgical	1	Congenital
	1	Colloid
	4	Adenomatous
	5	Toxic
	5	Thyroiditis
	4	Carcinoma
X-ray (in addition to surgery)	1	Thyroiditis
	3	Carcinoma

A summary of treatment carried out appears in Table II.

COMMENTS

This survey does not indicate the incidence of goiter among children in this community, as some go untreated and others are treated medically on an outpatient basis. It serves merely as a basis for discussion of the problem of childhood goiter and its management. It will be noted that just as in adults females are more commonly affected than males, the ratio in this series being about three girls to one boy, or even higher if malignant disease is included.

Why some babies are born with enlargement of the thyroid gland is not known. Occasionally cases have been attributed to ingestion of antithyroid drug by the mother [*6,8*], but more often no dietary, drug, geographic or hereditary factor can be held responsible. The gland in congenital goiter is characteristically soft, tends to encircle the trachea and presents a characteristic histologic appearance. The enlargement may disappear spontaneously in the

first few weeks of life. Thyroid extract may be effective in reducing the size of the gland. Iodine and x-ray therapy are useless [*11*]. Thyroidectomy may be required because of pressure symptoms or, as in our case, for cosmetic reasons as the child grows older.

Colloid goiter is more common than the two cases included in this report would indicate. This is particularly true in an endemic area such as the Northwest. However, in this day of diversified diet and iodized salt, cases requiring hospital care are seldom seen. The slight enlargement of the gland so often observed in adolescence usually regresses spontaneously. Some of the larger colloid goiters may be reduced in size by the administration of desiccated thyroid. Because of the soft nature of the gland local symptoms are uncommon. The basal metabolic rate is usually normal or slightly depressed. Surgical resection is seldom indicated unless adenomas are thought to be present, pressure symptoms occur or an unsightly deformity develops.

Adenomatous goiter is not and should not be considered to be a clinical diagnosis. If one excludes the children having thyrotoxicosis, all but four of our patients had a "nodular goiter" on examination. Following operation they were classified according to the pathologic process found. This is an exceedingly important point. The term nodular goiter does not denote a benign lesion nor is it synonymous with adenomatous goiter. This is amply illustrated by the fact that of fourteen patients having nodular goiters only four were found to have benign adenomas. Much time is wasted in futile polemics regarding the incidence of carcinoma in nodular goiter. Differences in statistical computations, classifications, pathologic interpretations and even semantics compound the confusion. It should suffice simply to know that a nodule is not necessarily a benign adenoma and that the possibility of carcinoma exists in order to justify its removal. This should far outweigh such specious arguments against thyroidectomy in childhood that adenomatous goiter is more apt to recur than if the patient is operated upon later in life or that the actual incidence of malignancy is low. The treatment of choice for multinodular goiter is radical subtotal thyroidectomy; for the solitary nodule, total lobectomy should be performed on the affected side [*16*]. Because of the high incidence of malignancy, conservative treatment with cytomel, thyroid extract, etc. has no place in the treatment of nodular goiter in children.

Toxic goiter is not a rare disease in childhood. Its onset often occurs in early adolescence, but may be earlier; its severity may be far greater than is estimated clinically although in general its manifestations are similar to those noted in adults. The disease occasionally regresses as the child grows older, but may become increasingly severe. Enlargement of the gland is usually diffuse [*12*] although it was nodular in two of this small group of seven patients. Because of the increased operative risk in children, conservative therapy with antithyroid drugs is the initial treatment of choice. However, if response is not prompt, complete and lasting, thyroidectomy is indicated. All too often one sees children who have been subjected to prolonged drug therapy during which they have never enjoyed really good health or developed normally, children who could have obtained prompt relief from a properly planned and executed operation. It is our opinion that subtotal thyroidectomy, after proper preparation and with careful postoperative supervision, is the treatment of choice in the majority of cases and will yield the best results and the lowest incidence of recurrence. As our experience indicates, the course is often stormy but need not be disastrous and an excellent ultimate outcome is to be anticipated.

Thyroiditis at one time was extremely rare in children [*12*], but a recent increase in its incidence has been noted [*7,11*]. We have seen five such patients. In none was the correct diagnosis considered preoperatively. Pathologic findings were characteristic of struma lymphomatosa in all patients. In all respects the disease is similar to that encountered in adults, even to the uniform tendency for hypothyroidism to develop in the postoperative period. One cannot but wonder what the future holds for these children in view of the observation of Dailey and his associates [*3*] that malignant neoplasms occur four times more often in thyroids with some degree of Hashimoto's thyroiditis than in those without it.

We were shocked to find four patients with carcinoma in this small group of twenty-three children hospitalized for treatment of goiter (20 per cent of those operated upon). As a check on this we investigated the next older age

group (fifteen through twenty years of age) at Sacred Heart Hospital over a five year period (1951 through 1955) and found that there were six patients with carcinoma of the thyroid among the thirty-two older children hospitalized for treatment of goiter. In the younger age group all were girls, in the older there were five girls and one boy. Statistics regarding the incidence of carcinoma will vary according to the manner in which they are compiled, i.e., whether one considers all goiters, nodular goiters, solitary nodules, or excludes cases diagnosable clinically or those in which no palpable abnormality of the thyroid exists. The percentage is unimportant. It is 100 per cent to the child who has carcinoma. What is important is the realization that carcinoma of the thyroid does occur with surprising frequency in children.

We can draw no conclusions regarding the proper mode of treatment from this small series. Published opinions vary from the ultraconservatism of Crile [*1*] to the more widely accepted radical approach consisting of total [*15*] or radical subtotal thyroidectomy and neck dissection. Perhaps the best advice is that of Ward [*18*] who suggests that "therapy must be individualized according to the pathologic picture, the possibility of complete extirpation, the presence of metastasis, the affinity of the tumor for radioactive iodine (I-131) and the sensitivity of the tumor to external radiation." Postoperatively it is important to maintain these children on thyroid extract. Although few deaths from cancer of the thyroid in children occur, a considerable number of these patients will ultimately succumb to their disease.

Recently some alarming reports have appeared [*2,4,14*] indicating the surprisingly large number of children with cancer of the thyroid who have previously received radiation therapy for benign diseases of the tonsils, adenoids, thymus and so on. Unfortunately we have no data concerning this in our cases, but the reported incidence is high enough to make coincidence seem unlikely and to suggest that radiation of the neck in children is potentially dangerous.

SUMMARY

A study of twenty-three children under fifteen years of age hospitalized for the treatment of goiter is reported. Four of these patients had papillary carcinoma of the thyroid gland. Treatment was by surgery and radiation. Tetany developed in one of these patients. Of the nineteen patients with benign disease all but three were treated surgically. Mild postoperative hypothyroidism was the only persistent complication encountered. There were no deaths.

REFERENCES

1. Crile, G., Jr. The pattern of metastasis of carcinoma of the thyroid. *Ann. Surg.*, 143: 580–587, 1956.
2. Clark, D. E. Association of irradiation with cancer of the thyroid in children and adolescents. *J. A. M. A.*, 159: 1007–1009, 1955.
3. Dailey, M. E., Lindsey, S. and Miller, E. R. Histologic lesions in the thyroid glands of patients receiving radioiodine for hyperthyroidism. *J. Clin. Endocrinol.*, 13: 1513–1524, 1953.
4. Duffy, B., Jr. and Fitzgerald, P. W. Cancer of the thyroid in children. *J. Clin. Endocrinol.*, 10: 1296–1309, 1950.
5. Harper, H. P., Durham, M. W. and Holm, J. C. Carcinoma in nodular goiter: incidence in the inland empire. *Northwest Med.*, 54: 37–39, 1955.
6. Handelsman, J. C. and Sussman, H. Suffocative goiter in the newborn. *Ann. Surg.*, 145: 108–114, 1945.
7. Hayles, A. B., Kennedy, R. L. J., Woolner, L. B. and Black, B. M. Nodular lesions of the thyroid gland in children. *J. Clin. Endocrinol.*, 16: 1880–1994, 1956.
8. Hepner, W. R., Jr. Thiourea derivatives and fetus: review and report of case. *Am. J. Obst. & Gynec.*, 63: 869–874, 1952.
9. Kunstadter, R. H. and Stein, A. F. Treatment of thyrotoxicosis in children with thiourea derivatives. *Pediatrics*, 6: 244–253, 1950.
10. McGavack, T. H. Treatment of hyperthyroidism. *M. Clin. North America*, 37: 695–703, 1953.
11. Norris, W. J. and Pollack, W. F. Nodular goiters in children. *Am. J. Surg.*, 90: 345–352, 1955.
12. Pemberton, J. J. and Black, B. M. Goiter in children. *Surgery*, 16: 756–763, 1944.
13. Riley, W. A. Quoted by Kunstadter, R. H. and Stein, A. F. [*9*].
14. Rienhoff, W. F. Discussion of paper by Crile, G., Jr. [*1*].
15. Ross, D. E. Cancer of the thyroid gland in children. *Surg., Gynec. & Obst.*, 104: 433–440, 1957.
16. Schnug, G. E. Surgical treatment of non-toxic adenomatous goiter. *West. J. Surg.*, 62: 335–337, 1954.
17. Searls, H. H. Quoted by Ward, R. [*18*].
18. Ward, R. Cancer of the thyroid in children. *Am. J. Surg.*, 90: 338–344, 1955.
19. Winship, T. Carcinoma of the thyroid in children. *Tr. Am. Goiter A.*, 11: 364–389, 1951.

The Chvostek Sign

A CLINICAL STUDY

ELMER HOFFMAN, M.D., *Baltimore, Maryland*

From the Departments of Surgery, The Johns Hopkins University School of Medicine and Hospital, and the Sinai Hospital of Baltimore, Inc., Baltimore, Maryland.

THE author first became especially interested in the Chvostek sign because of the large percentage of positive responses which he had found in the preoperative thyroid patients of the younger age group. The value of the sign in postoperative thyroidectomies for the prediction, or the follow-up, of the cases of tetany was lost in these patients. Therefore the knowledge of the percentage occurrence of the Chvostek sign in normal individuals would be useful. The study was undertaken with the idea of making basic, general observations on the clinical occurrence of the Chvostek sign.

Definition. The facial phenomenon which is called the Chvostek sign consists of the rapid contraction of the facial muscle (or muscles) supplied by the seventh cranial nerve, elicited on tapping the nerve itself or its branches. Chvostek [*1*] described the increased irritability of the facial nerve. He demonstrated this by percussing the nerve in front of the external auditory meatus (Chvostek I). Schultze [*2*] elicited Chvostek's sign by percussing a region between the zygomatic arch and the corner of the mouth (Chvostek II).

Mechanism. Various explanations have been put forward to explain the phenomenon. Van der Elst [*3,4*] claimed that the Chvostek sign was due to reflexes set up by deep receptors at the point stimulated. Others also thought that the Chvostek signs were reflexes [*5,6*]. Still others have believed that the Chvostek sign was due to the direct mechanical stimulation of the muscles [*7,8*].

Kugelberg [*9*], by timing the events of the Chvostek sign, showed that the sign is due to mechanical stimulation of motor fibers in the facial nerve when the percussion point is in front of the auditory meatus. When the area between the corner of the mouth and the zygoma was considered, it was often due to mechanical stimulation of the motor fibers, occasionally due to a reflex, and occasionally due to both. There was no evidence that the Chvostek sign was due to direct stimulation of the muscles.

Etiology. The Chvostek sign has been reported as being positive in many unrelated disease processes [*10,11*]. These include the following: tetany in the newborn, hypoparathyroidism, alkalosis, rickets, diphtheria, measles, scarlet fever, whooping cough, typhoid fever, tonsillar diseases, enteroptoses, myxedema, joint neuralgia and tuberculosis. The only common denominator which can be picked out is a lowered serum calcium level (particularly the ionized portion) in a small percentage of cases. However, even in low calcium, or hypoparathyroid, tetany, the Chvostek sign is not always positive. It has also been stated that the positive Chvostek sign in infectious diseases is a reflection of the increased reflex irritability accompanying the disease process. The Chvostek sign has also been positive in individuals without any known disease. Some observers have found the Chvostek sign to occur in a very irregular, unpredictable fashion, even disappearing and recurring in the same patient.

Incidence in the Literature. There is no exact method of production of the Chvostek sign reported in the literature. The author's method of production is described hereafter. There is also very little to be found in the world literature with respect to the significance of the occurrence of the Chvostek sign in certain age groups, racial groups or in terms of bilaterality

or repetition. The author will attempt to answer some of these problems a little later in this paper.

Ford [*12*] states that the Chvostek sign is of little significance during the first week of life and after the fifth year of life. He also points out that even a normal nerve will react to a hard blow. Kletz and Elmalek [*13*] found the sign positive in nineteen of twenty-six pairs of twins, with the only difference between twins being one of intensity or strength of response.

In 1951 Uhse [*10*] examined 425 children of all ages (but he does not give a breakdown of ages). He found that 198 or 45.4 per cent of 435 showed a positive Chvostek sign (and here he does not state whether he is referring to Chvostek I or Chvostek II). Of the 198 children, ninety-eight or 49.5 per cent were boys, and 100 or 50.5 per cent were girls. Uhse writes that in boys and girls the great majority of positive cases occur between the ages of six and ten years, and that the incidence of positive responses decreases from there on in a regular fashion. These observations differ from those made by the author.

Uhse [*10*] found that sixty-two children (thirty boys, thirty-two girls) of the 198 with positive signs showed a definite difference as to the side on which the Chvostek phenomenon could be elicited. Sixteen of the boys and twenty-one of the girls had a positive response only on the right side. The phenomenon was absent four times on the right side but could be elicited on the left. In the remaining cases the Chvostek sign remained weaker or stronger on one side or on the other. An explanation for unilaterality of response, or for the difference in strength of responses between the two sides, is difficult to find. Stieve (quoted by Uhse [*10*]) believes that there is no anatomical basis. Uhse raises the possibility of hemisphere dominance (there were no left-handed children in his study). Uhse also was unable to correlate the Chvostek response with the color of the hair, the type of hair, the color of the iris, the color of the skin or the disease diagnosis.

Bass (quoted by Uhse [*10*]) found a positive Chvostek sign in 20 per cent of the older school children.

Uhse also quotes Von Pfaundler as having noted that the Chvostek sign was positive ten times more frequently in patients seen in private practice than in ward or clinic patients.

Hummel [*14*] examined 4,000 pupils from all kinds of schools and found no sex difference in the occurrence of the positive Chvostek sign. He found a 70 per cent constancy in that the students were either permanently Chvostek-negative or Chvostek-positive (this group was made up of all the students). Hummel [*14*] and Uhse [*11*] suggest the existence of an age disposition for the manifestation or disappearance of the Chvostek sign as well as an individual disposition. Hummel [*14*] states that 30 per cent of 2,497 elementary school students, 19 per cent of 1,063 grade school students and 40 per cent of 954 high school students showed a positive Chvostek sign. Hummel found no correlation between the presence of a positive Chvostek and the social strata of the students.

Bell and Bartels [*15*] quote an incidence of 5 per cent positive Chvostek tests in the normal population.

MATERIAL AND METHODS

The author has attempted to record the following features of the Chvostek phenomenon: (1) constant method of production; (2) incidence of positive response in normal individuals; (3) incidence of positive response in various age groups; (4) incidence of positive response in racial groups; (5) incidence of positive response in sex groups; (6) incidence of positive laterality (absence on one side and strength of one side over the other); (7) incidence of positive Chvostek I and/or II responses; (8) constancy of positive or negative response; (9) familial tendency to the positive Chvostek sign; (10) correlation of positive response with blood chemical studies; (11) correlation of positive response with diagnosis of patient.

The selection of cases was carried out in such a way as to eliminate those patients who already had a disease state known to be associated with the presence of tetany, such as hypoparathyroidism, hypocalcemia, uremia, etc. Thus it might be stated that the patients in this study were normal patients or patients with illnesses not associated with hypocalcemia. In a certain percentage of the positive cases blood calcium levels (as well as other determinations) were carried out. When it became obvious that there was no definite correlation, these tests were not continued as a routine measure. The cost and the amount of laboratory personnel time involved outweighed the value of the negative information gained.

Method of Production. In reviewing the literature the author found no detailed description of the method of eliciting the Chvostek sign. In an effort to carry out a study which could be compared with others, I have used the following repeatable method. The Chvostek I

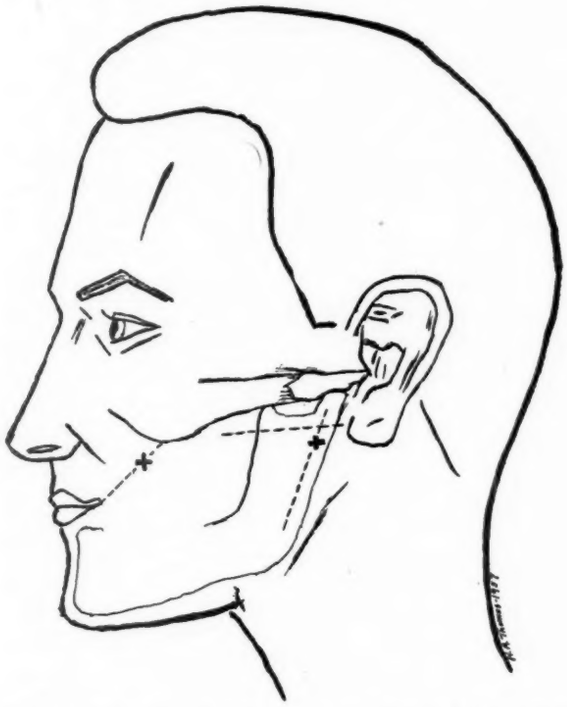


FIG. 1. Location of points for eliciting the Chvostek signs.

phenomenon is elicited by *light* percussion over a point located as follows: (1) 0.5 to 1 cm. below the zygomatic process of the temporal bone; (2) 2 cm. anterior to the ear lobe; (3) on a line with the angle of the mandible. The Chvostek II phenomenon is elicited by light percussion over a point located on the line joining the zygomatic prominence and the corner of the mouth and about one-third of the distance from the zygoma. Figure 1 illustrates these points of location. In the individual with an elongated mandibular body and prominent or protruding lips the Chvostek I point is more anterior, and the Chvostek II spot is closer to the zygomatic arch.

A word of warning is offered against repeated firm percussion quite close together in time relationship (that is, taps within seconds of each other). This may cause a positive response to become negative temporarily. However, a 15 to 30 second rest, followed by light percussion, will again reveal a positive response.

For the most part the positive Chvostek sign did not involve the mandibular or ophthalmic branches (in all but five instances tested).

Occasionally the jar of the tap will cause

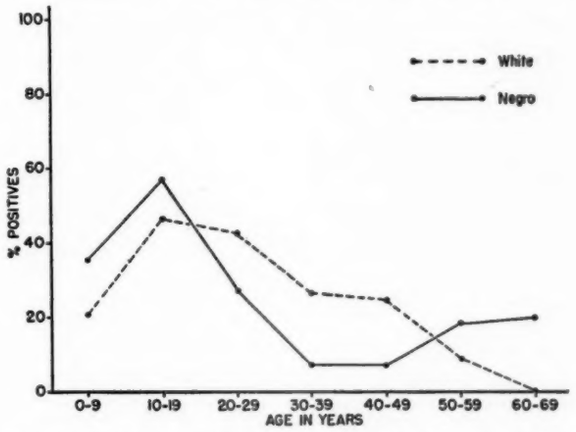


FIG. 2. Incidence of positive Chvostek signs in males.

motion of the angle of the lip and the nose, and thus give the mistaken impression of a positive Chvostek sign. Lipstick applied widely at the corners of the mouth sometimes obscures the positive response. If the masseter muscle is kept tense, there may be a false negative response. Relaxation of the masseter may then yield a positive response.

RESULTS

The author has tested 736 normal patients (both clinic and private, white and Negro, male and female) for the presence or absence of a positive Chvostek sign.

Age, Race and Sex Distribution. Figures 2 and 3 are graphic representations of the per cent positive Chvostek signs found in the various age groups and sexes. There was a 20.4 per cent incidence of a positive Chvostek sign in the zero to nine year age group of white males. In the ten to nineteen year age group it was 46.6 per cent; twenty to twenty-nine years, 42.5 per cent; thirty to thirty-nine years, 27.4 per cent; forty to forty-nine years, 24.6 per cent; fifty to fifty-nine years, 8.5 per cent; sixty years and over, 0 per cent. The figures in the Negro males of corresponding age groups were: 35.7, 57.1, 27.2, 7.1, 7.6 and 18.1 per cent. In the Negro males of sixty to sixty-nine years, the incidence was 20 per cent. There were no positive Chvostek signs in Negro males of seventy years or over. The over-all

incidence of the positive Chvostek in white males was 24.2 per cent; in Negro males, 21.7 per cent.

There was a 28.5 per cent incidence of a positive Chvostek sign in the zero to nine year age group of white females. In the ten to

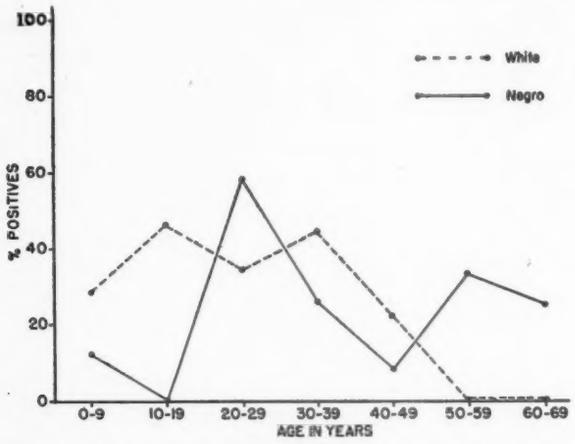


FIG. 3. Incidence of positive Chvostek signs in females.

nineteen year age group it was 47 per cent; twenty to twenty-nine years, 34.6 per cent; thirty to thirty-nine years, 44.5 per cent; forty to forty-nine years, 22.4 per cent. There were no positive signs in the patients fifty years of age and over. In Negro females the figures for the incidence of a positive Chvostek sign in the corresponding age groups were: zero to nine years, 12.5 per cent; ten to nineteen years, 0 per cent; twenty to twenty-nine years, 58.3 per cent; thirty to thirty-nine years, 26 per cent; forty to forty-nine years, 8.3 per cent; fifty to fifty-nine years, 33.3 per cent; sixty to sixty-nine years, 25 per cent; seventy years and over, 0 per cent. The over-all incidence of the positive Chvostek sign in white females was 28.3 per cent; in the Negro females, 24.6 per cent.

In the total population tested there was a 24.7 per cent occurrence of a positive Chvostek sign.

Laterality of Response. The Chvostek sign was found to be positive on the left side in 7.3 per cent of those patients in whom the response was elicited. The Chvostek sign was positive on the right side in 11 per cent of the patients with this sign. The Chvostek sign could be elicited on both the left and the right sides in 80.5 per cent of the patients in whom the sign was positive. There was no definite correlation with age, sex or race.

The positive Chvostek sign was stronger on the left side than on the right in 3.6 per cent of the total number of positive cases, and vice versa in 5.7 per cent of the cases.

Chvostek I and II. The positive Chvostek signs in this study had the following distribution: the Chvostek I was present in 4.7 per cent of the positive cases, the Chvostek II in 23.7 per cent, and the Chvostek I and II in 71.6 per cent of the cases.

Constancy of Response. Although the follow-up examinations of the patients with positive Chvostek signs were limited in number, there was an indication that once a patient exhibited a positive Chvostek response, it remained so. However, there might be a variation in the laterality of the sign or in the intensity of the response.

Familial Tendency. The author's data on the familial incidence of the positive Chvostek are not statistically significant because of inadequate numbers, but there is an indication of a tendency for a positive sign to be present in the parent when it is present in the offspring. This is not necessarily true in the reverse direction; that is, an offspring need not exhibit a positive Chvostek sign simply because his parent has one.

Blood Chemistry. As stated earlier, blood samples from the first twenty-five patients exhibiting a positive Chvostek sign were analyzed for the level of urea, calcium, phosphorous, albumin, globulin and alkaline phosphatase with negative correlative results. The same was true of urinary Sulkowitch testings. Because of the laboratory expense and the personnel time involved, as well as the lack of positive correlative findings, this phase of the project was discontinued.

Disease Process Present. This series of cases revealed no common denominator in terms of a specific disease process being present or absent.

CONCLUSIONS

The incidence of the positive Chvostek signs in 736 patients tested was 24.7 per cent (the incidence in white males was 24.2 per cent, in Negro males 21.7 per cent, in white females 28.3 per cent, and in Negro females 24.6 per cent). The ten to nineteen year age group was the group showing the age peak incidence in white males, Negro males and white females. The twenty to twenty-nine year age group

showed the age peak incidence in Negro females. There is a greater chance of occurrence of a positive Chvostek sign in the older age groups of the Negro than of the white. There was no positive Chvostek sign in any white person over the age of sixty years or in any Negro over the age of seventy years.

In 80.5 per cent of the cases in which the Chvostek sign was positive it could be elicited on both the left and right sides. Both the Chvostek I and II signs could be elicited in 71.6 per cent of the positive cases. There is an indication of the constancy of the positive Chvostek sign in the individual case. There is also some indication of a familial incidence of the positive Chvostek, with the parent usually showing a positive response if the child has one.

No correlation was demonstrated between the occurrence of a positive Chvostek sign and blood or urinary changes or the presence of specific disease processes.

SUMMARY

The occurrence of the positive Chvostek sign is analyzed in terms of age, sex, race, laterality, persistence of sign, familial tendency and blood chemistry values.

REFERENCES

1. Chvostek, F. Beitrag zur Tetanie. *Wien. med. Presse*, 17: 1201, 1876; 20: 1201, 1879.
2. Schultze, F. Über Tetanie und die mechanische Erregbarkiet der Nerven. *Deutsche. med. Wchnschr.*, 8: 276, 1882.
3. Van der Elst, P. R. Over het verschijnsel van Chvostek en den Gelaatreflex, Inaug. Dissert., Amsterdam, 1928.
4. Van der Elst, P. R. Das Chvosteksche Phänomen. *Jabrb. f. Kinderb.*, 123: 288, 1929.
5. Pavel, I., Claudian, J. and Cornateano, D. La significance clinique et physio-pathologique du signe de Chvostek. *Ann. méd.*, 27: 86, 1930.
6. Weingrow, S. M. Facial reflexes. *Arch. Pediat.*, 50: 234, 1933.
7. Parhon, C. L. and Schachter, M. Paralysis faciale périphérique et signe de Chvostek. *Bull. et mém. Soc. d'endocrinol.*, p. 31, 1937.
8. Schachter, M. Paralysie faciale périphérique et signe de Chvostek. *Praxis*, 35: 542, 1946.
9. Kugelberg, E. The mechanism of the Chvostek sign. *Arch. Neurol. & Psychiat.*, 65: 511–517, 1951.
10. Uhse, W. Das Facialisphänomen. Das Deutsche Gesundheitswesen. *Ztschr. für Med.*, 49: 1405–1436, 1951.
11. Wechsler, I. Textbook of Clinical Neurology. Philadelphia, 1952. W. B. Saunders Co.
12. Ford, F. Diseases of the Nervous System in Infancy, Childhood and Adolescence, 3rd ed. Springfield, Ill., 1952. Charles C Thomas.
13. Kletz, H. P. and Elmalek, H. Study of the Chvostek sign in twenty-six pairs of twins. *Ann. endocrinol.*, 12: 4, 1951.
14. Hummel, H. Spätfacialis und Schularzt. Das Deutsche Gesundheitswesen. *Ztschr. für Med.*, 28: 873–905, 1952.
15. Bell, G. O. and Bartels, E. Postoperative parathyroid tetany. *Labey Clin. Bull.*, 7: 105–109, 1951.

Restoration of the Thoracic Esophagus with Aortic Homograft Following Palliative Subtotal Esophagectomy

FRANK A. ROGERS, M.D., *Whittier, California*

From the Department of Surgery, College of Medical Evangelists School of Medicine, Los Angeles, California.

CARCINOMA of the esophagus accounts for 2 per cent of all deaths due to cancer and continues to be diagnosed in a relatively advanced stage of the disease. Many patients reach the operating room in an incurable if not inoperable state. The reasons for the poorer prognosis in this type of cancer have been previously outlined.

Because of the many resections for carcinoma that are purely palliative and the extensive surgery necessary to remove the primary tumor and then re-establish continuity by esophagogastrostomy or esophagojejunostomy, many attempts have been made to invent lesser operative procedures aimed at palliation. Although helpful in some instances, the majority of experiences with such procedures have been discouraging [*1,2,9*].

Several years ago Souttar [*3,4*] discussed palliative dilatation of the esophagus. He described insertion of a coiled silver wire which could be pushed down the esophagus after serial dilatations and thus aid in delaying closure of the lumen by tumor growth. He added a further note on intraluminal radiation by suspending a narrow cylinder containing radon gas from a hook on the upper spiral of the indwelling esophageal wire. Souttar, however, completely despaired at removal of any esophageal carcinoma because of the depth of the organ, the fragile nature of the esophageal wall, the complexity of the adjacent structures and the advanced age of most of the patients.

Carter [*5*] has more recently advocated a palliative procedure similar to that of Souttar, consisting of forceful dilatation through the tumor and endoscopic insertion of a rigid tube prosthesis in order to maintain patency of the esophagus. In nineteen cases treated by this method between 1950 and 1956 there were five perforations of the esophagus and four deaths. Thirteen patients were able to swallow liquids and semi-solids for one to twelve months.

Mackler and Mayer [*6*] also have discussed palliation of esophageal obstruction due to carcinoma and have reported their experiences with a plastic intraluminal tube placed directly into the esophagus after intrathoracic esophagotomy. The tubes used measured about 12 cm. in length with a diameter of 0.9 to 1.2 cm. Such a tube is placed by threading it over a Levin tube brought out through a longitudinal incision in the esophagus above the tumor. A flange on the end of the tube rests on the upper margin of the tumor. In nineteen trials on eighteen patients there were six operative deaths (31.5 per cent). Of twelve survivors, five succumbed within an average of 3.4 months, while seven others had lived an average of four months at the time of reporting.

Berman [*7,8*] has offered a palliative approach to neoplastic occlusion of the esophagus consisting of resection of the tumor and replacement of the defect by means of a polyethylene tube. The plastic tubes, which are sutured to cuffs of the transected ends of esophagus, are essentially non-reactive. Some spill of food has occurred around the upper ends of the tubes and leakage has been common. The use of the Berman tube does involve direct resection of the tumor, and results in a mortality rate no higher than other procedures which are only attempts to maintain a narrow lumen in the involved segment of esophagus.

An ideal palliative procedure should be one which prolongs life, allows the patient to swallow soft food and has a low mortality rate. Such a procedure has as yet not been found, but the search should continue for two reasons. First, because the survival period for patients with untreated cancer of the esophagus is so short. Wu and Loucks [9] have pointed out that survival following cases of esophageal carcinoma that prove to be non-resectable is about 1.7 months. These authors found an average survival period of only 2.2 months after gastrostomy procedures. Second, it is now obvious that even the most aggressive extirpative attack on this type of malignancy results in little more than poor palliation. Of a collected series of 2,500 cases of carcinoma of the esophagus surveyed by Mackler and Mayer there were only 28 five-year survivors. Furthermore, the operative resectability in this group was only 35 per cent and the gross mortality averaged 40 per cent.

The results with surgical excision followed by radiation therapy, while occasionally resulting in a long term cure, have generally been disappointing.

With this general analysis in mind, it would seem that for the majority of patients with cancer of the thoracic esophagus the best palliative procedure would be removal of the primary tumor and reconstruction of the esophagus with a tubular substance that could be carefully anastomosed to the divided ends of the esophagus. The use of preserved human arterial homografts may offer some advantages in re-establishing continuity in the esophagus after subtotal resection for carcinoma.

The caliber of the average adult thoracic aorta is suitable for anastomosis to the divided ends of the esophagus and provides a very adequate lumen for the passage of swallowed food. The preserved thoracic aortic segments are available and are in much less demand than are the bifurcation aortic grafts. When reconstituted, the freeze-dried homografts present a firm yet elastic edge for suture anastomosis and their insertion is known to be followed by a fairly rapid production of fibrous tissue around the graft. Long segments are available so that it is possible not only to resect the esophageal tumor but also to include a fair amount of esophagus above and below the lesion and thus anastomose it to ends of the esophagus which would be free of tumor.

Another possible advantage of this type of prosthesis is that the primary tumor is removed. Only a thoracotomy is needed and the patient is thus spared the more severe thoracolaparotomy or two-team thoracotomy and laparotomy procedures.

The use of aortic grafts is not suggested for any case in which there is hope of cure by more adequate resection of the esophagus and regional lymph nodes. It may be useful, however, in the purely palliative epidermoid carcinoma of the esophagus when lymph nodes are involved and there has been obvious penetration to mediastinal tissues.

The advisability of postoperative deep x-ray therapy in the form of supervoltage machines or radioactive cobalt is at present being evaluated. It would seem, however, that should postoperative radiation be used, a graft such as the arterial homograft would withstand the inevitable deep tissue reaction as well or better than some other form of living tissue bridge.

A case is reported to illustrate the use of an aortic homograft for palliative resection of carcinoma of the esophagus.

CASE REPORT

A. W., a sixty-five year old Caucasian man, was admitted to Murphy Memorial Hospital on March 7, 1957. He complained of severe dysphagia with partial inability to swallow even liquids. His dysphagia had been progressive over the previous two and a half months. He had lost approximately 8 pounds and had noted difficulty first with thicker, more solid food. Gradually he had become unable to swallow satisfactorily any food including water. There had been no vomiting, hematemesis, cough or thoracic pain. He reported some burning in the epigastric area approximately two years prior to his present illness which became asymptomatic in the latter months of 1956 on antacid therapy. He noted that his food would seem to stick at a level high in the chest. He had had no previous operations and denied any serious illnesses or injuries. A review of systems was not contributory and neither the history nor physical examination revealed any evidence of disease or involvement of any system other than the gastrointestinal system.

Laboratory analysis including urinalysis, hemogram and blood chemistries were essentially normal. A chest film was reported as being within normal limits. X-rays of the esophagus revealed a widening of the cervical esophagus above a lesion which narrowed and constricted the upper portion of the thoracic esophagus. A normal distal thoracic esophagus was seen from the mid-portion down-

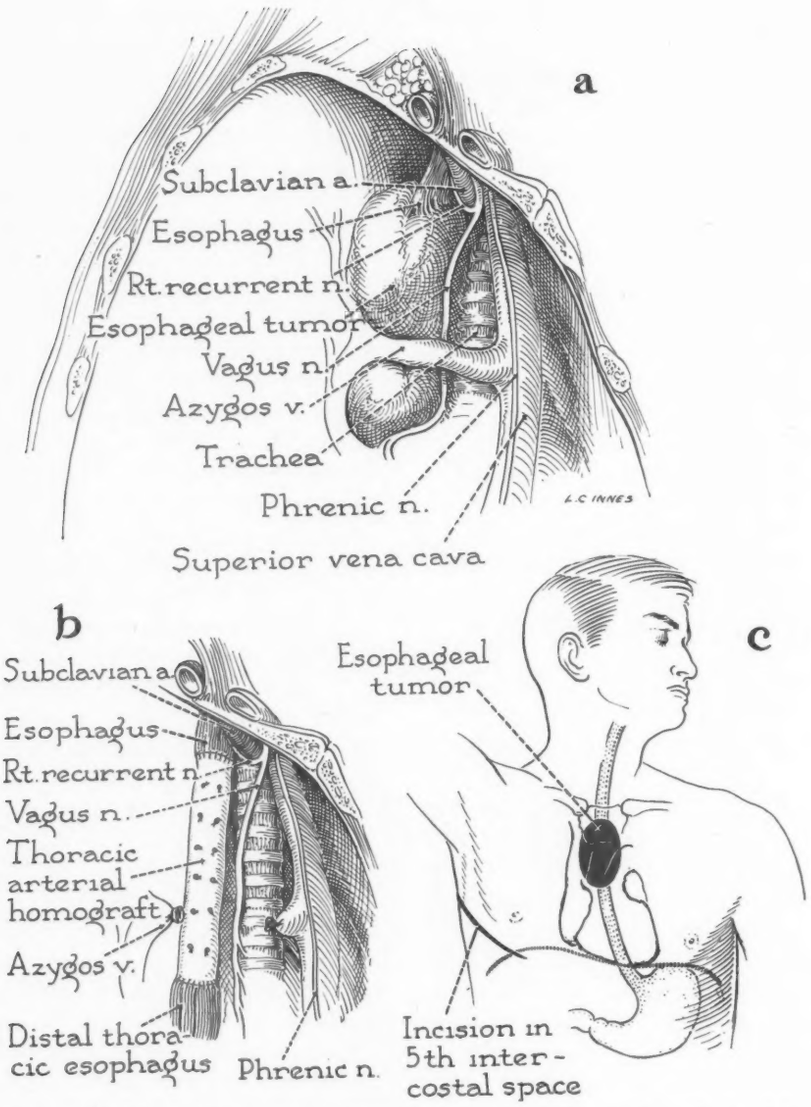


FIG. 1. (*a*) View of large tumor of upper thoracic esophagus through the right chest. (*b*) A preserved thoracic aortic homograft has been sutured to the divided ends of the esophagus following palliative resection of the tumor. (*c*) The location and extent of the tumor are diagrammed and the site of incision is indicated.

ward. The stomach and duodenum were not remarkable. On March 11th bronchoscopy and esophagoscopy were carried out in the operating room. An 8 by 40 bronchoscope was passed with ease and revealed the trachea and main stem bronchi to be intact. The posterior and left lateral wall of the trachea bowed forward and to the right, and some rigidity was noted. Esophagoscopy revealed constriction of the lumen blocking the passage of the esophagoscope at a level just past the thoracic inlet. Here a nodular, somewhat reddened tumor was noted and biopsies were taken which revealed the presence of epidermoid carcinoma. The patient was scheduled for surgery.

Following induction the patient was carried on an ether-oxygen mixture given through an endotracheal tube. The upper thoracic and abdominal areas were prepared with pHisoHex® followed by tincture of zephiran,® and sterile drapes were applied to expose the right thoracic area first. The right side of the chest was entered through a posterolateral incision carried through the fifth intercostal space. (Fig. 1*c*.) The right lung was completely adherent to the parietal peritoneum by moderately firm adhesions, preumably due to an old pleural effusion. There was a firm nodular area in the medial segment of the right middle lobe. The adhesions between the lung and the parietal

pleura and mediastinal pleura were all separated and the lung was allowed to collapse. An extremely large, nodular tumor occupied the upper thoracic esophagus. (Fig. 1*a*.) There was adherence of the tumor to the trachea anteriorly and extension to the mediastinal pleura immediately overlying the tumor. There was no free fluid within the thoracic cavity and no evidence of any pleural spread. Three slightly enlarged, firm nodes located on the lateral aspect of the upper portion of the tumor were found to contain carcinoma. Two large, hard nodules were also palpated subsequently in the left upper mediastinum. The gross palpable portion of the malignancy terminated at the thoracic inlet above and the lower level extended down a distance of 15 cm. to mid-thoracic esophagus. The tumor measured 7 cm. in greatest diameter.

The azygos vein was ligated and doubly tied with No. 0 silk. The mediastinal pleura over the tumor was excised widely and the upper end of the tumor was freed by sharp and blunt dissection. A portion of the cervical esophagus was pulled down and the recurrent laryngeal nerve identified and spared. The vagus nerve was divided subsequently as it curved sharply posteriorly over the lower portion of the tumor just above the hilus of the lung. The gross tumor and adjacent lymph nodes were freed after dividing the esophagus approximately 2 cm. above its gross extent toward the neck. A large Atraugrip clamp was used to hold the proximal esophageal segment after dividing above the tumor. Dissection was carried downward and the esophagus transected at the level of the seventh thoracic vertebra.

A segment of preserved human thoracic aorta was then removed from a vacuum tube and reconstituted in saline containing terramycin.® The intercostal branches were tied with No. 0000 silk and the graft substituted for the defect in the esophagus. (Fig. 1*b*.) The anastomoses were completed with a single layer of interrupted No. 0000 silk sutures.

The right lung was re-expanded and the previously noted nodule in the middle lobe was removed by a wide wedge resection removing a segment of lung which was roughly triangular and measured 5 to 6 cm. along the base. This pulmonary defect was closed with interrupted mattress stitches of No. 000 chromic catgut. The chest was closed in layers and drained with two catheters attached to a sealed underwater drainage bottle.

The patient's position was then changed slightly toward the supine and a short incision was made through the epigastric area through the linea alba fascia. The lower portion of the stomach was identified and a No. 18 Robinson catheter, with additional openings cut in the distal end, was placed through a stab wound in the antral area. This was fixed in position with three successive purse string sutures of No. 00 catgut after the method of Stamm. The catheter was brought out through a separate stab incision in the left epigastric area and the abdomen was closed with interrupted cotton sutures.

The patient withstood the surgery unusually well. His postoperative course was remarkably free from difficulties and he was able to begin gastrostomy feedings with similac within forty-eight hours. He coughed well, was able to ambulate early and was discharged from the hospital on the seventh postoperative day.

He continued his gastrostomy feedings at home for another week. At the end of this time he was given a soft diet which he tolerated well and reported no swallowing difficulty. Because of the known residual nodal tumor in the upper posterior mediastinal area, the patient was referred for Tumor Board consultation and deep x-ray therapy.

Postoperative x-ray examination revealed a marked narrowing at the area of the distal anastomosis which was thought to be due to residual tumor. Esophagoscopy with dilatation was carried out approximately one month postoperatively.

The patient was last seen on December 24, 1957, at which time bronchoscopy and esophagoscopy were done. Recurrence of tumor in the proximal cervical esophagus was present and completely blocked the esophageal lumen. This was presumed to be recurrence at the proximal suture line. There was further evidence of extension of the tumor with concentric narrowing of the proximal trachea. The patient had maintained his weight to this time, but now continues to be fed totally by gastrostomy tube.

SUMMARY

A case of esophageal carcinoma treated by palliative resection and replacement of the portion of the thoracic esophagus with an arterial homograft is presented. This method of esophageal resection presents an easy and reasonable palliative procedure. Preserved human aortic segments give an adequate lumen, and early sealing at the anastomotic sites is believed to occur. The use of gastrostomy feedings in the immediate postoperative period is advantageous in alimenting the patient for a period while fibrotic healing of the anastomoses is occurring. Since there is a natural tendency for stenosis to occur at the suture lines, normal swallowing should be resumed as soon as a reasonable time has elapsed for sealing of the anastomoses. A careful single layer anastomosis allows for minimal concentric narrowing of the esophageal lumen. An effort should be made even in palliative

resection to resect beyond areas of intramural carcinomatous extension.

Further clinical use of aortic homografts for esophageal replacement after palliative resection seems worthwhile.

REFERENCES

1. Ravitch, M. M. and Bathson, H. T. Carcinoma of the esophagus; a consideration of curative and palliative procedures. *J. Thoracic Surg.*, 24: 256–265, 1952.
2. Allison, P. R. and Borrie, J. The treatment of malignant obstruction of the cardia. *Brit. J. Surg.*, 37: 1–21, 1949.
3. Souttar, H. S. The treatment of carcinoma of the esophagus. *Brit. J. Surg.*, 15: 76–94, 1927.
4. Souttar, H. S. Carcinoma of the esophagus. *Brit. M. J.*, 2: 797–800, 1934.
5. Carter, M. G. Endoscopic intubation and dilatation in the management of esophageal carcinoma. *Am. J. Surg.*, 93: 266–270, 1957.
6. Mackler, S. A. and Mayer, R. M. Palliation of esophageal obstruction due to carcinoma with a permanent intra-luminal tube. *J. Thoracic Surg.*, 28: 431–445, 1954.
7. Berman, E. F. The experimental replacement of portions of the esophagus by a plastic tube. *Ann. Surg.*, 135: 338–343, 1952.
8. Berman, E. F. The plastic esophagus. *J. Internat. Coll. Surgeons*, 18: 695–699, 1952.
9. Wu, Y. K, and Loucks, H. H. Carcinoma of the esophagus and cardia of the stomach. *Ann. Surg.*, 134: 946–956, 1951.

"Endoaneurysmorrhaphy, or the Matas operation, is a derivative of the Antyllian operation. It was first performed by Matas in 1888. The procedure is applicable to all aneurysms in which the circulation through the sac can be provisionally controlled. The principle of the operation rests upon the fact that the aneurysmal sac, which is lined by more or less modified intima, when not disturbed from its vascular connections, is capable of exhibiting reparative reaction similar to that possessed by like structures elsewhere. The circulation is arrested by means of a tourniquet or, when this is impracticable, by exposure and compression of the main artery on each side of the aneurysm. The sac is then exposed by a free incision parallel with its long axis. It is important that the attachments of the sac to the surrounding tissues remain undisturbed. The sac is then opened in the axis of the artery from one extremity to the other and cleared of its contents, the interior of the sac being gently but thoroughly scrubbed with gauze in order to remove adherent laminated blood clots. According to the type of sac present one may then proceed to perform an obliterative, restorative, or reconstructive operation." (From Bryant, J. D. and Buck, A. H. American Practice of Surgery. Baltimore, 1910. William Wood & Co., division of Williams & Wilkins Co.)

Ruptured Pericostal Steel Wire Retention Sutures

A POST-THORACOTOMY COMPLICATION

WALTER H. MASSION, M.D., MICHAEL T. LATEGOLA, PH.D. AND JOHN A. SCHILLING, M.D.,
Oklahoma City, Oklahoma

From the Departments of Surgery and Physiology, the University of Rochester School of Medicine and Dentistry, Rochester, New York and the University of Oklahoma School of Medicine, Oklahoma City, Oklahoma.

IN the course of a study of physiological and morphological effects of lung resection, reported elsewhere [1], a large number of thoracotomies were performed in dogs. The immediate postoperative recovery period was uneventful in all animals, yet a serious and totally unexpected complication, the rupture of pericostal steel retention sutures, occurred frequently after five to ten days, and was fatal in three dogs. This report is written to call attention to the possible occurrence of similar events and their sequelae in human thoracic surgery.

OPERATIVE METHODS

Mongrel dogs weighing between 20 and 30 kg. were used. Each animal was anesthetized with nembutal,® placed on its side and connected to a mechanical respirator by means of an intratracheal tube. The chest was opened through a liberal seventh rib interspace incision. After lobectomy was performed, the chest wall was reapproximated by two No. 25 stainless steel wire, buried, pericostal retention sutures. Great care was taken to avoid any kinking of the wire and no excessive tension was employed when the ends were twisted together. Four layers of sutures were used to complete closure of the chest wall, a continuous No. 3-0 silk suture for the intercostal muscles, interrupted No. 3-0 silk for the serratus and pectoralis muscles, interrupted subcutaneous No. 3-0 silk and a continuous No. 3-0 silk subcuticular suture for the skin. Strict aseptic precautions were observed. The animals received 600,000 units of penicillin and one gm. of streptomycin daily for four days following the operation. The physiological measurements were taken under nembutal anesthetic with the dog placed in the supine position.

CLINICAL SEQUELAE OF RUPTURED WIRE RETENTION SUTURES

In four of eight dogs various complications occurred after the first postoperative week which could all be traced to the same origin. These are described in the following protocols.

DOG 56-22 (22 kg.). The right lower, middle and mediastinal lobes were resected. On the seventh postoperative day an extensive subcutaneous emphysema was observed, which was very pronounced in the head and neck region, and extended back to the lower portions of the hind legs. On both sides of the incision in the chest the skin was tightly inflated and bulged to an extent that rupture of the wound seemed imminent. (Fig. 1.) Despite his monstrous appearance the animal did not appear clinically ill. Spontaneous activity and appetite remained normal and the dog recovered completely in the course of five weeks. It was sacrificed 100 days after operation.

On the ninth postoperative day, a positive pressure pneumothorax of $+0.2/-5.0$ cm. H_2O was present on the operated side which refilled rapidly after suction treatment. The total O_2 consumption was 115 ml./min. and the total tidal volume was 248 ml. On the 100th postoperative day the intrapleural pressure was $-8.0/-11.0$ cm. H_2O. The total O_2 consumption was 294 ml./min., and the total tidal volume was 360 ml. The static total-chest

FIG. 1. Dog 56-22, in supine position, chest and abdomen partly shaved. The skin is tensely inflated by a subcutaneous emphysema. The operative scar (arrow) appears to be buried by the bulging skin (seventh postoperative day).

compliance at a pressure of 20 cm. H_2O was 68.3 ml./cm. H_2O and this is 100 per cent of the preoperative control value.

Postmortem examination revealed that the remaining right upper lobe was covered with thickened pleura and scar tissue. It was atelectatic with the exception of the apex and had a liver-like appearance. The cut surface of the parenchyma had a greenish-yellow color. The wire retention sutures were broken and covered with fibrotic tissue.

DOG 56-26 (36 kg.). The right lower, middle and mediastinal lobes were resected in the first operation. The left upper and medial lobes were removed fifty days later. Recovery after the first operation was normal except for a slight loss of weight (2.5 kg.). After the second operation loss of weight continued, the weight being 29.5 kg and 27.7 kg. on the eighth and thirty-sixth postoperative days, respectively. Stridor could be noticed during moderate exercise. The dog died suddenly during the night of the thirty-sixth postoperative day after it had been placed on its left side in its cage to recover from light nembutal anesthesia induced for a study period.

The intrapleural pressure on the thirty-sixth day was $-4.0/-6.0$ cm. H_2O. The O_2 consumption of the right lung was 65 ml./min. and on the operated left lung 91.5 ml./min. The tidal volumes were 236 ml. and 171 ml., respectively.

On postmortem examination the left side of the chest cage was full of blood, the source of bleeding being the heart, which revealed a rent in the left ventricle through which a 5 mm. lead pencil could be introduced. The pericardium was filled with blood and clots. The left lower lobe was grossly normal. The steel wire retention sutures in the operative scar were ruptured, with a 6 mm. long fragment protruding into the intrapleural space. This appeared to have punctured the pericardium and myocardium with fatal tamponade.

Examination of the right side of the chest revealed the upper lobe to be adherent to the operative scar in the chest wall for a length of about 4 cm., but otherwise normal. Dissection of the scar in the chest wall revealed two broken wire sutures, their fragments being well covered by and imbedded in scar tissue without penetrating into the intrapleural space.

DOG 55-454 (19 kg.). The right lower, middle and mediastinal lobes were resected in the first operation. The left upper and medial lobes were removed forty-six days later. Immediate postoperative recovery after both procedures was uneventful. Eight days after the second operation the dog showed signs of respiratory distress; his tongue was cyanotic and the animal had to lie down frequently during a short walk. An emergency operation was performed on the same day; the left lower lobe was removed because of the extensive pathologic condition secondary to laceration by a 7 mm. long fragment of broken steel wire which protruded into the intrapleural cavity at the site of the lung injury. The dog died six hours after this operation.

On the eighth day after the second operation the intrapleural pressure was $+1.0/-2.0$ cm. H_2O and the pneumothorax refilled rapidly after aspiration. The O_2 consumption on the right side was 52.8 ml./min., the tidal volume 133 ml.; on the left side we found an O_2 consumption of 46.5 ml./min. BTPS and a tidal volume of 59 ml.

The operative specimen revealed that the left lower lobe was atelectatic and fibrotic, being about one-third of its normal volume. The anterior surface showed deep, fresh, as well as old lacerations, with thickened pleura and scar tissue covering the lobe. A leakage of air could be demonstrated by inflating the lobe from its segmental brochus.

On postmortem examination the left side of the chest was empty except for a small amount of serosanguineous fluid. Examination of the

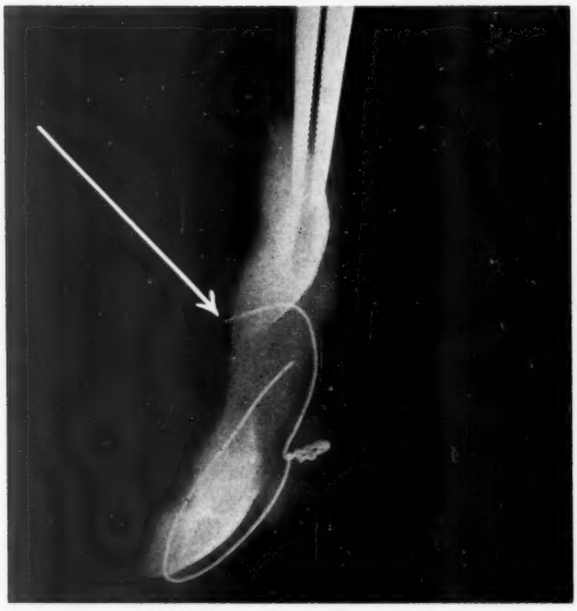


FIG. 2. Dog 56-25. X-ray film of a specimen from the chest wall. The penetrating end of the broken pericostal wire (arrow) has lacerated the left lower lobe (Fig. 3), causing the death of the animal. Note that the wire is broken in the ring portion and not where the ends were twisted together.

FIG. 3. Dog 56-25. Postmortem lung specimen of left lower lobe, dried by air inflation. The traumatic damage by the broken wire retention suture is readily observed.

right side of the chest revealed a normal appearing upper lobe except for a small area which adhered to the anterior chest wall. Broken but fibrotically exteriorized wires were found in the old scar in the chest wall. The trachea was filled with bloody foam. The right ventricle and atrium were enlarged.

DOG 56-25 (31 kg.). The right lower, middle and mediastinal lobes were removed in the first operation. The left upper and medial lobes were resected fifty-nine days later. Immediate postoperative recovery was uneventful. On the eleventh day after the second operation the dog exhibited signs of respiratory distress. A moderate emphysema of the skin could be palpated, which was localized around the operative incision extending down to the inguinal region. The dog died during the night of the eleventh postoperative day.

The intrapleural pressure on the right side was +6.0/0.0 cm. H_2O. The O_2 consumption on the right side was 66.5 ml./min., the tidal volume 68 ml. On the left side the respective values were 74 ml./min. and 63 ml. After aspiration of approximately 500 ml. of air from the intrapleural space, the lung function improved. The intrapleural pressure was now −3.0/−6.0 cm. H_2O. The O_2 consumption on the right side was 61 ml./min., the tidal volume 99 ml.; on the left side O_2 consumption was 106 ml./min. and the tidal volume 113 ml. The static chest compliance was 21.3 ml./cm. H_2O at an airway pressure of 20 cm. H_2O. This represents a decrease of 32.6 per cent if the last measurement before the second operation is taken as 100 per cent.

Postmortem examination revealed that the wire suture in the left chest wall was broken and the sharp edges had scratched the lower lobe. (Fig. 2.) The lobe was partly atelectatic and showed extensive traumatic damage. (Fig. 3.) Several air leaks could be demonstrated. The remaining right upper lobe was grossly normal. Two broken wires were found in the right chest wall; they were covered with scar tissue.

COMMENTS

Airtight stable closure of the chest cage is essential for the function of the lung after thoracotomy. Since the chest wall cannot be temporarily immobilized as most other regions of the body, the closure of the chest presents particular problems. Strong costal retention sutures are often used to hold the neighboring ribs of the intercostal incision firmly together. Because of its strength and tissue compatibility, steel wire would seem to be particularly suited to achieve this effect although silk or

chromic sutures are more commonly employed. The early repeated occurrence of wire rupture and its sequelae, however, were not anticipated.

High quality stainless steel wire can be exposed momentarily to very high tension before breakage occurs [2]. It may be assumed that the frequent although small strain exerted by the inspiratory expansion of the chest produced early occurrence of metal fatigue or hysteresis. The intermittent tension together with the biochemical environment of the tissues may have produced a phenomenon which is well known to metallurgists as "stress-corrosion" [3]. A simple experiment showed that the wire had undergone a change in its physical properties in spite of a spotless external appearance. When a new wire and a wire which had been in the body for five days were repeatedly bent 180 degrees, the wire recovered from the operative scar broke after thirty bendings whereas the new wire withstood 112 bendings before breakage occurred.

The great vulnerability of the intrathoracic organs makes the occurrence of a wire rupture a serious hazard. The danger may be somewhat greater in the dog with a more compressible thorax and higher respiratory frequency, nevertheless it also exists in the human being if such materials and technics are employed. The events that occurred in this small experimental series of animals have been recorded to call attention to the stress-corrosion phenomenon of stainless steel wire as it may be employed in human clinical surgery.

SUMMARY

Pericostal stainless steel retention sutures employed in the closure of thoracotomies in dogs showed a high incidence of breakage. This led to various postoperative complications, three of which were fatal. The stress-corrosion phenomenon, i.e., the combination of tensile strain and biochemical corrosion, is considered responsible for the early rupture of the stainless steel sutures. The use of this material in thoracic surgery seems contraindicated.

REFERENCES

1. Lategola, M. T., Massion, W. H. and Schilling, J. A. Physiological and morphological effects of lung resection in dogs. USAF School of Aviation Medicine, Report No. 57–65.
2. Thum, E. E. The Book of Stainless Steels. Cleveland, 1935. American Society for Metals.
3. Metals Handbook. Cleveland, 1948. American Society for Metals.

Carcinoma of the Colon and Rectum in Persons under Twenty Years of Age

M. Tischer Hoerner, M.D., *Dayton, Ohio*

There is increasing evidence to show that age is no barrier to the occurrence of carcinoma of the colon and rectum in young persons. It has been generally appreciated that sarcomas are common in children, but epithelial malignancies have been found in sufficient instances to warrant their consideration in a differential diagnosis of childhood diseases. Rankin and Graham [*44*] state that the incidence of carcinoma of the large intestine in young persons is probably proportionally higher than the incidence of carcinoma invading all organs of the body. Williams [*57*] reviewed reports of 1,934 cases of carcinoma in general and found 0.99 per cent in persons thirty years of age or less. Rankin and Comfort [*43*], in reviewing 1,452 patients with carcinoma of the rectum, found an incidence in the same age group of 3.85 per cent. Pennington's [*41*] study of 7,313 collected cases of carcinoma of the large intestine seemed to substantiate the aforementioned figure, for he reported an incidence of 3.7 per cent in persons below thirty years of age.

Carcinoma of the colon and rectum in young patients below twenty years of age, while not common, began to be discovered and scattered reports appeared in the literature. Walker and Daly [*53*] reviewed the literature in 1934. They found ten proved instances of carcinoma of the colon in children under fifteen years of age. Reports of cases were also made on this subject by Pfeiffer and Wood [*42*], Ogilvie [*38*] and Warthen [*54*]. In 1943 Bacon [*1*] reported ninety-four cases in the literature of carcinoma of the rectum in patients under twenty years of age. Johnston [*25*] collected all the cases available in 1947 and found forty-eight instances of carcinoma of the large intestine, excluding the rectum and rectosigmoid, in persons aged less than seventeen years.

In the same year that Johnston [*25*] made his report, Bonelli [*6*] reviewed the literature for all malignant tumors of the small intestine and large intestine in young people through the age of nineteen years. Besides the patients listed by Johnston, he found two more with malignant lesions of the descending colon, four with similar lesions in the sigmoid, and twelve unreported incidences of carcinoma of the rectosigmoid in youths in this age group. In addition, Bonelli [*6*] found reports of seventy cases of carcinoma of the rectum. All references were rechecked but it was impossible to determine to what extent the cases listed by Bonelli [*6*] overlapped with those recorded by Bacon [*1*] and others previously. Under these circumstances it was believed best to include all of the seventy cases of carcinoma of the rectum reported in the publication by Bonelli [*6*] so that none would be overlooked.

The literature has been reviewed and brought up to date in this paper. Eighteen additional cases of carcinoma of the colon in young persons under twenty years of age have been found. Twelve more instances of carcinoma of the rectum have been recorded in youths in the same age group. (Table 1.)

One youth suffering from carcinoma of the sigmoid colon and another having a malignant growth in the rectum are reviewed in this article and added to the literature. These new additions, when added to those obtained from all sources mentioned previously, make a grand total of 189 cases of carcinoma of the rectum and seventy-three instances of malignant disease of the colon in patients under twenty years of age. Rankin and Graham [*44*] and Williams [*58*] state that in children, as in adults, carcinoma of the lower sigmoid and rectum is three times as common as in other segments of the colon. While it is true that malignant

disease is seen more often in the lower portions of the large intestine in children, the cases reported in the literature suggest that the ratio between cancerous growths at this site and those situated at higher levels of the large bowel should be approximately 2.5:1.

TABLE 1

Author, Year Reported, Ref. No.	Carcinoma of Colon (No. of Cases)	Carcinoma of Rectum and Rectosigmoid (No. of Cases)
Goden, 1877 [21]	..	1
Dunbar, 1879 [14]	1	..
Barber, 1919 [4]	1	..
Rosenberg, 1934 [46]	3	1
Bottin, 1935 [7]	..	1
Netto, 1935 [34]	1	..
Jordan and Chamberlin, 1937 [26]	..	1
Mayo and Madding, 1940 [31]	..	1
Oosting, 1941 [39]	..	2
Scholefield, 1946 [50]	1	..
Saner, 1946 [47]	1	..
Fishback, 1947 [18]	1	..
Wilkerson, 1947 [56]	..	1
Bellone, 1947 [5]	1	..
Kerr, 1948 [27]	1	..
Marion and Avet, 1948 [29]	..	1
Nelson et al., 1949 [33]	1	..
Mass. Genl. Hosp., 1949 [30]	..	1
Read, 1950 [45]	1	..
Chauvenet et al., 1952 [10]	1	..
Newns and Donald, 1952 [35]	1	..
Gross, 1953 [23]	..	1
Schilla, 1954 [49]	..	1
Williams, 1954 [57]	3	..
Total	18	12

CASE REPORTS

CASE 1 E. W., an eighteen year old boy, began to notice a small amount of blood in his stool in January, 1950. It was red in color and observed once or twice a week until February, 1950. In March he began to have mild pain across the lower abdomen intermittently. The pain would last for a few hours at a time and was "crampy" in nature. The passage of flatus or a stool would relieve the discomfort. The latter part of March a small amount of blood was observed again in the stool occasionally. The abdominal pain became worse in April, 1950, and began to be present after the ingestion of his meals. No nausea or vomiting was noted but the patient lost his appetite and even became "afraid to eat" because of the distress that followed. No diarrhea was noted but constipation became progressively more marked until a cathartic was required for the passage of a stool.

On examination on April 6, 1950, the patient was found to be a well developed and nourished white male. No pathologic disorder was found related to the head, neck or thorax. The systolic blood pressure was 116 and the diastolic 78. Very slight tenderness was detected on palpation in the left lower abdomen. There was no rigidity present and no masses were felt. The genitalia were normal. No ulceration or masses in the rectum were discovered. His extremities revealed no abnormal findings.

Proctoscopic examination was performed and no pathological lesion detected. X-ray of the colon revealed a partially obstructing lesion, suggestive of a malignant neoplasm, present in the sigmoid colon. There were no significant findings in his blood studies other than the presence of a mild secondary anemia. Examination of the urine showed no abnormalities.

A diagnosis of carcinoma of the sigmoid producing partial large bowel obstruction was made. The colon was decompressed by conservative methods. Nothing was given by mouth for two days; fluids were administered intravenously. Warm colonic irrigations were used with the patient in the knee-chest position. When results from the irrigations were obtained, a liquid diet was given by mouth. Small doses of Fleet's phospho-soda® began to be tolerated and assisted in the passage of daily stools. After the edema of the bowel at the site of partial obstruction subsided, the patient could assimilate a low residue diet. The colon was cleansed by means of chemotherapy and the glycogen stored in the liver replenished by daily intravenous injections of glucose.

After adequate preparation the patient was operated upon on April 25, 1950. Carcinoma of the sigmoid colon was detected and resected. Intestinal continuity was re-established by means of end-to-end anastomosis.

The pathological report revealed that the gross specimen consisted of a segment of colon 18 cm. in length. About 6 cm. from the lower edge of the bowel was a neoplastic growth, measuring 4 cm. in diameter, which encircled two-thirds of the circumference of the wall. There was central ulceration. The edges were indurated, and the regional lymph nodes were enlarged.

Histologic examination of sections from the colon showed a highly malignant adenocarcinoma. There was considerable invasion of the muscularis. Many adenomatous masses of tumor tissue were seen invading the muscularis and extending along the submucosa. In some areas the tumor formed solid

cords of neoplastic cells showing marked anaplasia. One lymph node was almost completely replaced by metastatic process.

The patient had an uneventful convalescence and was dismissed from the hospital on May 8, 1950, on a low residue diet. On returning home he apparently was so hungry that he "overate" and was not careful of his diet. On May 10, 1950, mild abdominal pain developed. On May 11th numerous small stools were passed and slight rectal bleeding was noted. Pain was noted on defecation. The patient was readmitted to the hospital and fluids were administered intravenously. After cleansing the colon of accumulated feces, liquids and finally solid foods were tolerated by mouth. He was again discharged from the hospital in good condition on May 15, 1950.

The patient has remained in good health to date. He was followed up four times a year for three years. Since then he has been checked yearly. These examinations have included six-month and finally yearly x-ray studies of the colon. No pathologic disease has been detected.

CASE II. R. D., a nineteen year old boy, began to observe slight rectal bleeding intermittently in October, 1954. This stopped after being present for three weeks. In November, 1954, rectal soreness was felt on defecation and vague pelvic discomfort began to be noted at times. The pelvic pain extended across the lower abdomen and became much worse in December, 1954. Constipation developed the same month. The patient had lost 10 pounds in weight since October, 1954. No loss of strength was reported. Prior to the origin of these complaints the patient had always been in good health.

When the patient was examined on December 2, 1954, he was found to be a well developed, well nourished white male. No abnormal findings were detected in the head, neck or thorax. The abdomen was not distended. No enlargement of the liver was detected. There was mild tenderness on palpation in both lower quadrants of the abdomen, more marked in the left lower abdomen. No rigidity or masses were detected, and no hernias were present. The genitalia were normal. On rectal examination an indurated, elevated irregular lesion could be felt at the tip of the examining finger. The mass was large and extended beyond the range of palpation. Ulceration was detected in the lesion. Proctoscopic examination revealed the same massive, ulcerated lesion of the rectum, originating 7 cm. above the anus. The upper portion of the neoplasm could not be detected for the tumor involved the entire circumference of the rectum and partial obstruction existed at the upper limits of the tumor. A biopsy specimen was taken and the neoplasm was reported to be an adenocarcinoma.

It was doubted that the lesion was resectable but it was believed that abdominal exploration should be performed after thorough preparation of the colon. Exploration was carried out through a low left rectus incision on December 10, 1954. The carcinoma of the rectum was found to extend upward to the rectosigmoid, and the entire floor of the pelvis was studded with minute, grayish white, elevated, indurated nodules. These lesions caused marked thickening and some edema of the pelvic peritoneum, as well as the tissues behind the bladder. There was enlargement of the pelvic lymph nodes and many of the mesenteric and abdominal nodes, including those along the aorta. There was no definite evidence of metastasis to the liver. One of the lymph nodes along the aorta, as well as a section of the pelvic peritoneum, was removed for biopsy purposes. These tissues were reported to be adenocarcinoma. As the neoplasm was not resectable, a permanent type of loop colostomy was made employing the sigmoid colon. It was believed that this procedure would give palliative relief for the impending obstruction of the rectum.

The patient recovered from the operative procedure. However, after four months' freedom from symptoms, evidence of widespread metastasis was detected. Subsequently, his course went progressively downward and he succumbed to the disease in July, 1955, approximately nine months after the first symptoms developed.

PATHOLOGY

When considering the characteristics of malignant disease of the large intestine, it is best to divide this organ into right and left halves. Neoplasms in the right half of the colon in children usually present a different picture from those in the left. Carcinoma of the right half of the colon often is composed of a large, polypoid, fungating lesion which may grow to considerable proportions before producing symptoms. Right-sided neoplasms usually do not produce obstruction because: (1) they are not likely to encircle the colon; (2) the bowel content of this portion of the intestine is liquid; and (3) there is a tendency for the lesions to perforate and form abscesses.

Malignant growths on the left side of the colon in children are prone to produce various degrees of obstruction in the colon relatively early in the course of the disease. Several factors are involved in the process: (1) the diameter of the colon is narrower on the left side; (2) the fecal content of the bowel tends to be formed; and (3) many of the neoplasms tend to circle the colon in a signet-ring manner.

Malignant epithelial tumors of the large intestine are usually one of three types of lesions: (1) adenocarcinoma, probably the most common; (2) scirrhous carcinoma, the type that is seen so frequently in left-sided lesions of the colon and is responsible for the development of obstructive phenomena; and (3) mucoid adenocarcinoma or mucoid carcinoma which constitutes about 5 per cent of all neoplasms of the colon and rectum in adults. It is interesting to note that in children mucoid carcinoma is found in 50 per cent of the patients having malignant disease of the large intestine.

Sarcomatous growths of the large intestine are reported occasionally. Galloway and Owens [*20*] discovered seven cases of sarcoma of the colon in children in the literature and added one of their own. Bonelli [*6*] found reports of thirteen cases of sarcoma of the large intestine and rectum in young people. Bacon, Wolfe and Archambault [*2*], and Erbes and Stehlin [*15*] added individual cases of their own. A survey of the literature suggests that epithelial tumors of the large intestine are more common in young persons. However, Bonelli [*6*] believes that quite the opposite is true as far as the small intestine is concerned. He found sixty-three cases of sarcoma of the small intestine with 77.6 per cent of the lesions being located in the ileum. There were only twelve instances of malignant epithelial disease of the upper intestinal tract.

SIGNS AND SYMPTOMS

The symptoms of carcinoma of the colon in children are not specific. Probably because of the nature of the nerve supply of the large intestine and by virtue of its size, as to width of lumen, as well as territory in the abdomen covered, pain when present often is not at the site of the lesion. This is especially true of the so-called "silent" right colon, for symptoms frequently are not noted until considerable alteration in structure or function has occurred.

The pain associated with a lesion in the right colon is frequently referred to the epigastrium. The pain or distress may be cramplike or aching in character. The mild dyspepsia that is sometimes seen in association with recurrent appendicitis is also sometimes observed in these patients. There are reports in the literature [*33,58*] in which the patient was operated upon for appendicitis and the appendix was not found to be causing the illness. Subsequently, the etiology of the pain was discovered to be carcinoma of the colon. Such observations emphasize the importance of making a thorough exploration of the abdomen before closure of the wound when a child is operated upon for chronic, recurring attacks of abdominal pain and the cause is not apparent. Instead of explaining the abdominal pain or enlargement of the mesenteric lymph nodes on the basis of mesenteric adenitis, consideration should always be given to the possibility of the presence of carcinoma of the colon in children.

Not infrequently the first sign of disease of the right colon will be the accidental discovery of a mass in this area. Due to the size of the lumen of the bowel, the tumor may reach considerable proportions before producing symptoms.

Anemia is another cardinal sign of malignant disease of the right colon. It may vary from a mild secondary anemia to an anemia so profound that the patient is unable to be as active as formerly.

As has been suggested, when malignant neoplasms involve the left colon, they usually present the characteristic picture that one ordinarily associates with carcinoma of the large intestine. In contradistinction to malignant growths in the right colon, those on the left in children often afford more evidence of their location. It is not difficult to understand the reason for the change in the presenting symptoms if one remembers that: (1) the diameter of the colon is narrower in this portion of the intestine; (2) the fecal content tends to be more formed; and (3) the neoplasms at this site are prone to encircle the bowel. As a result of these phenomena, obstruction is the outstanding characteristic of lesions in this side of the colon. Irregularity of the stool may develop early in the course of the disease. As the growth becomes larger, there may be alternating constipation with abdominal cramping and urgency, and diarrhea with blood in the stool. The symptoms are usually intermittent. However, obstruction is not uncommonly complete during the first attack in children and it may be the initial indication that there is something wrong in the colon.

Malignant growths of the rectosigmoid and rectum in young persons are probably easier to diagnose than neoplasms anywhere else in the

large intestine, for the symptoms, while not characteristic, are quite suggestive. All that is needed to confirm the diagnosis is a careful digital examination, or at most a proctoscopic examination. Although pain is not always present and often is mild, it is usually located in the left side of the abdomen or in the pelvis. Bleeding is not common in children with carcinoma of the colon. However, when the malignant lesion involves the rectum, rectal bleeding is seen more frequently. Lesions at the rectosigmoid are apt to produce obstructive features but those in the ampulla of the rectum are likely to be silent for a longer period because the lumen of the bowel is wider at this point.

TREATMENT

The treatment of choice for all malignant lesions of the large intestine in children is radical resection of the growth and restoration of the continuity of the bowel by immediate anastomosis. The incidence of high grade malignancy and the frequency with which early metastasis is discovered in children with carcinoma of the large intestine makes extensive resection an essential feature in the treatment of the condition. Resection and primary anastomosis in the presence of marked obstruction is contraindicated. Consequently, there are instances when decompression of the bowel is essential and a two-stage operation is a life-saving and proper procedure.

COMMENTS

The prognosis of carcinoma of the colon and rectum in children is not good. The longest periods of survival of young people afflicted with this disease found in the literature are the cases of Webster [*55*] and Scholefield [*50*]. The former reported a patient who lived four years after operation, and the latter a youth who showed no demonstrable recurrence seven years after surgery was performed. The first patient reported in this paper has lived over eight years following resection of the malignant growth in the colon. This constitutes possibly the longest survival on record. When one has to resort to reporting success in the treatment of individual patients, it emphasizes the fact that the therapy administered to most patients suffering from this disease leaves much to be desired.

One reason for the poor prognosis is due to failure to diagnose and treat children with neoplasms of the bowel early. Many men do not appreciate that malignancy of the colon can occur at an early age. The youngest patient with this disease reported in the literature [*12*] was three years and three months old. Many of the patients operated upon were not properly prepared for surgery of the colon because the preoperative diagnosis was often appendicitis, tuberculous peritonitis or acute intestinal obstruction. The fact that the symptoms of this condition in children are of relatively short duration adds to the difficulty of making an early diagnosis. It is well known that there is an increased incidence of early metastasis associated with the high grade malignancies so frequently seen in tumors of the large intestine in children. Until more reliable methods are suggested, careful consideration must be given to the milder complaints associated with disturbances of the intestine. It is only by being alert to the possibility of the presence of carcinoma of the colon in children that an early diagnosis can be made, and this is essential if we hope to save more of these patients.

So far we have only considered the highly malignant primary carcinomas of the large intestine in children. However, mention should be made of a second group of malignant lesions composed of those instances in which an adenomatous polyp of the colon undergoes malignant transformation. This subject has been thoroughly discussed by Snyder, Chaffin and Snyder [*52*]. These lesions can be treated conservatively and prophylactically. Benign polyps of the bowel often bleed before malignancy develops. Whenever bleeding from the rectum occurs in children, a thorough search should be made for the cause. The removal of a benign polyp may prevent the development of serious malignant disease of the bowel. Furthermore, it has been demonstrated that carcinoma of the large intestine in adult life can be due to malignant changes in polyps which manifested themselves in infancy or childhood.

SUMMARY

Age is no barrier to the occurrence of carcinoma of the colon and rectum in children. To date 189 cases of carcinoma of the rectum and seventy-three instances of malignant disease of the colon in patients under twenty years of age are recorded in the literature. These totals include one new case of each type reported in this paper.

Carcinomas of the right side of the colon in children do not tend to produce obstruction as do malignant lesions involving the left side of the large intestine. Mucoid adenocarcinoma constitutes approximately 50 per cent of all neoplasms of the colon and rectum in children. This type of tumor of the large bowel is found in only 5 per cent of adults suffering from malignant disease of the colon.

The symptoms of malignant disease of the colon in children are not specific and tend to differ from those found in adults. Pain is the most common symptom of the disease in young people. It may simulate that manifested by many abdominal conditions. For this reason, whenever a child is operated upon and the cause of the illness is not readily apparent, the possibility of the presence of carcinoma of the colon must be kept in mind. The accidental discovery of a mass in the right side of the abdomen or the presence of anemia may be the first indication that something is wrong with the right colon. Malignant tumors of the left colon often cause obstructive symptoms. Rectal bleeding is seen more frequently when a neoplasm involves the rectum.

The prognosis of carcinoma of the colon in children is poor. This is due to failure to diagnose and treat the condition early and because the lesions tend to be highly malignant and metastasize quickly. Early, radical resection of these growths offers the only hope for saving these young people. Removal of benign polyps of the colon may prevent the development of malignant transformation during childhood or adult life.

BIBLIOGRAPHY

1. Bacon, H. E. Anus, Rectum, Sigmoid Colon, pp. 562–566. Philadelphia, 1943. J. B. Lippincott Co.
2. Bacon, H. E., Wolfe, F. D. and Archambault, R. A. Rectal malignant tumor in childhood. *Am. J. Dis. Child.*, 64: 70, 1942.
3. Baillis, J. Cancer of rectum in child eleven years old; case. *Mém. Acad. chir.*, 68: 354, 1942.
4. Barber, W. H. Intestinal obstruction caused by adenocarcinoma. *Interstate M. J.* 26: 106, 1919.
5. Bellone, A. Carcinoma of sigmoid colon in nineteen year old boy; case. *Boll. e mém. Soc. toscoumbra chir.*, 9: 107–113, 1947–1948.
6. Bonelli, W. R. Malignant tumors of the small and large intestine in infants and children. *Clin. Proc. Child. Hosp.*, 3: 151–163, 1947.
7. Bottin, J. Une stinose rectale de diagnostic etiologique malaise. *Ann. Soc. méd.-chir. de Liége*, pp. 168–174, 1935.
8. Campos Rey de Castro, J. Neoplasms in children; study of 922 cases. *Arch. pediat., Uruguay*, 26: 816–827, 1955.
9. Chatterjee, J. Cancer as a pediatric problem. *Indian J. Pediat.*, 22: 208–214, 1955.
10. Chauvenet, A., Bouvard, A. and Berge, C. Carcinoma of the colon. *Arch. mal. app. digest.*, 41: 551, 1952.
11. Church, R. E. and Schwartz, A. D. Polyps of rectum and colon in children. *J. Pediat.*, 44: 104–111, 1954.
12. Clar. Quoted by Nothnagel, H. [36].
13. Daregon, H. W. Cancer in children from birth to fourteen years of age. *J. A. M. A.*, 136: 459–468, 1948.
14. Dunbar, L. Quoted by Bacon, H. E. [1].
15. Erbes, J. and Stehlin, J. S., Jr. Lymphosarcoma of the cecum with intussusception in a boy aged nine. *Am. J. Dis. Child.*, 85: 551, 1953.
16. Fanfani, M. Cancer in children; general data and specific anatomical sites. *Arch. "de Vecchi" per l'anat. patol. e la med. clin.*, 23: 149–388, 1955.
17. Firzli, S. Malignant tumors in early life. *J. Med. Libanais*, 8: 13–26, 1955.
18. Fishback, R. Carcinoma of transverse colon in fifteen year old boy; case report with short summary of literature. *Radiology*, 48: 168–171, 1947.
19. Fowler, L. H. Malignant epithelial neoplasms; carcinoma and epithelioma occurring in persons under twenty-six years of age. *Surg., Gynec. & Obst.*, 43: 73–84, 1926.
20. Galloway, W. H. and Owens, E. J. Primary lymphosarcoma of appendix occurring in childhood. *Brit. M. J.*, 2: 1387, 1949.
21. Goden. Quoted by Molliere, D. Traite des maladies du rectum et de l'anus, p. 565. Paris, 1877.
22. Goldstein, H. I. Malignant tumors of gastro-intestinal tract in young. *Schweiz. med. Wchnschr.*, 80: 1033–1035, 1950.
23. Gross, R. E. The Surgery of Infancy and Childhood, pp. 370–371. Philadelphia, 1953. W. B. Saunders Co.
24. Handy, V. H. and Golaberg, J. D. The occurrence of malignancies in childhood. *New York State J. Med.*, 56: 258–260, 1956.
25. Johnston, J. H. Carcinoma of colon in childhood and adolescence. *Am. J. Surg.*, 73: 703–712, 1947.
26. Jordan, S. M. and Chamberlin, D. T. Cancer of digestive tract in the young. *S. Clin. North America*, 17: 815–820, 1937.
27. Kerr, J. G. Polyposis of the colon in children. *Am. J. Surg.*, 76: 667–671, 1948.
28. Macaulay, D. and Steward, J. K. Malignant disease in childhood. *Med. Illus.*, 10: 284–290, 1956.
29. Marion, J. and Avet, J. Malignant degeneration of rectal polyp in child of four years. *Rev. mens. pediat., Lyons*, pp. 584–585, 1948.
30. Massachusetts General Hospital Case 35451: Colloid carcinoma of rectum with metastasis to regional lymph nodes in sixteen year old boy. *New England J. Med.* 241: 751–753, 1949.
31. Mayo, C. W. and Madding, G. F. Carcinoma of the rectum and of the rectosigmoid in the young. *Arch. Surg.*, 40: 83, 1940.
32. McFadden, H. W., Jr. Malignant tumors of child-

hood—pathology. *Nebraska M. J.*, 39: 375–378, 1954.

33. Nelson, C. W., Barr, R. M. and Deeb, P. H. Carcinoma of the colon in children. *Am. J. Surg.*, 78: 531–533, 1949.
34. Netto. Quoted by Jordan, S. M. and Chamberlin, D. T. [26].
35. Newns, G. and Donald, C. Case of carcinoma of colon in child. *Great Ormond St. J.*, pp. 64–67, 1952.
36. Nothnagel, H. Diseases of Intestines and Peritoneum, p. 402. Philadelphia, 1904. W. B. Saunders Co.
37. Ogauesyau, A. S. Cancer of rectum in children. *Pediatriya*, pp. 64–65, 1948.
38. Ogilvie, R. F. Mucoid carcinoma of caecum in boy of thirteen years. *Brit. J. Surg.*, 23: 601–606, 1936.
39. Oosting, M. Adenocarcinoma of sigmoid colon, rectum, and anus in children; report of two cases in thirteen year old girl and eight year old boy with summary of recorded cases up to fifteen years of age. *Obio State M. J.*, 37: 1067, 1941.
40. Paterson, E. Malignant disease in childhood. *South African M. J.*, 29: 1199–1206, 1955.
41. Pennington, J. R. Diseases and Injuries of the Rectum, Anus, and Pelvic Colon. Philadelphia, 1923. P. Blakiston's Son & Co.
42. Pfeiffer, D. B. and Wood, J. K. W. Cancer of transverse colon in seven year old boy. *J. A. M. A.*, 104: 1227–1229, 1935.
43. Rankin, F. W. and Comfort, M. W. Carcinoma of the rectum in young persons. *J. Tennessee M. A.*, 22: 37–42, 1929.
44. Rankin, F. W. and Graham, A. S. Cancer of the Colon and Rectum, 2nd ed., pp. 48–49. Springfield, Ill., 1939. Charles C Thomas.
45. Read, F. A. Carcinoma of splenic flexure in youth; case. *Connecticut M. J.*, 14: 105–108, 1950.
46. Rosenberg, H. Carcinom im jugendlichen Alter. *Ztschr. Krebsforsch.*, 41: 230–238, 1934.
47. Saner, F. D. Carcinoma coli in a child; case. *Brit. J. Surg.*, 33: 398–400, 1946.
48. Scaglione, G. Statistical data on the incidence of malignant neoplasms in children. *Arch. med. int.*, 7: 127–137, 1955.
49. Schilla, F. W. Carcinoma in a rectal polyp; report of case in infancy. *Am. J. Surg.*, 88: 759–760, 1954.
50. Scholefield, J. Carcinoma of colon causing acute intestinal obstruction in youth of seventeen. *Brit. M. J.*, 2: 461, 1946.
51. Sirsat, M. V. Cancer in infancy and childhood (from birth to fourteen years). *Indian J. M. Res.*, 41: 277–284, 1953.
52. Snyder, W. H., Jr., Chaffin, L. and Snyder, M. H. Neoplasms of the colon and rectum in infants and children. *Pediat. Clin. North America*, pp. 93–111, 1956.
53. Walker, R., and Daly, J. F. Carcinoma of upper colon in childhood. *J. Oklahoma M. A.*, 27: 119–121, 1934.
54. Warthen, H. J. Carcinoma of colon in childhood. Report of case. *Virginia M. Month.*, 64: 140–142, 1937.
55. Webster, R. Carcinoma of the colon in a child. *M. J. Australia*, 2: 907, 1938.
56. Wilkerson, V. H. Carcinoma of rectum in youth, with case report. *J. Internat. Coll. Surgeons*, 10: 414–419, 1947.
57. Williams. Quoted by Rankin, F. W. and Graham, A. S. [44].
58. Williams, C. Carcinoma of the colon in childhood. *Ann. Surg.*, 139: 816–822, 1954.

Adenocarcinoma of the Small Intestine

ALLAN E. GILBERT, M.D. AND ROBERT A. WISE, M.D., *Portland, Oregon*

From the Surgical Service, Veterans Administration Hospital, Portland, Oregon.

PRIMARY adenocarcinoma is the most common malignant tumor of the small intestine, but represents only a small percentage of gastrointestinal malignancy [*17,19*]. According to Anderson [*1*], 3 per cent of gastrointestinal carcinomas arise in the small bowel. Pridgen and co-workers [*15*], in a review of the literature, found the reported incidence to vary between 0.29 and 6 per cent, with an incidence of 1 per cent at the Mayo Clinic.

The experience of the Surgical Service of the Portland Veterans Administration Hospital was recently reviewed in a study of the Tumor Board files since 1934. Three cases of primary adenocarcinoma of the small intestine, representing an incidence of 0.35 per cent of gastrointestinal malignancy, were analyzed. In the first case the tumor was discovered at autopsy; a preoperative diagnosis of obstruction of the jejunum due to tumor was made in the second patient, and in the third patient the diagnosis was made at the time of surgery performed because of clinical and radiographic evidence of a perforated viscus with peritonitis. Each case represents a different complication of carcinoma of the small bowel and for this reason is considered worthy of presentation.

CASE REPORTS

CASE 1. A fifty-four year old white man was admitted to the Portland Veterans Administration Hospital on September 7, 1950 with the chief complaint of constant upper abdominal pain of ten months' duration. The pain was aggravated by the ingestion of food and relieved by vomiting or the passage of stool. The present illness was associated with a 10 pound weight loss, but no anorexia, hematemesis, melena or jaundice. The patient had been treated at a private hospital three months previously for a myocardial infarction and at that time had been given an ulcer regimen because of epigastric pain and anemia; no improvement had been noted.

Physical examination revealed a well nourished, pale white man whose only pertinent physical finding was tenderness between the umbilicus and right costal margin. No abdominal organs or masses were palpable and rectal examination was negative.

Laboratory data were as follows: The red blood cell count was 3.12 million per cu. mm.; the white blood cell count was 6,600 per cu. mm.; urinalysis was normal. Gastric analysis following an Ewald meal showed a total hydrochloric acid of 62 degrees and free hydrochloric acid of 26 degrees; two stool specimens were positive for occult blood.

Radiographic examination including an upper gastrointestinal series, oral cholecystogram and barium enema was reported as negative except for a single opaque stone in the gallbladder. An electrocardiogram was interpreted as abnormal, with myocardial irritability and ischemia of the posterior wall.

The patient was placed on strict ulcer management of bland diet, milk and antacids while undergoing evaluation. Immediately following the cholecystogram he complained of sudden substernal distress and nausea not relieved by nitroglycerin and oxygen; he died thirty minutes later.

At autopsy the immediate cause of death was found to be coronary occlusion and myocardial infarction. An incidental finding was a lesion of the duodenum 14 cm. from the pylorus at the junction of the second and third portions. (Fig. 1.) The mass, which measured 9 by 6 by 6 cm., nearly encircled the lumen and extended through the bowel wall into the root of the mesentery. The intrinsic part consisted of a necrotic ulcer crater, the base of which was covered with dark material suggestive of recent hemorrhage. The extraluminal portion extended into the root of the mesentery and pancreas, but no evidence of metastasis to regional lymph nodes or liver was found. Sections of the lesion revealed a transition from normal mucosa to neoplastic tissue composed of pleomorphic, hyperchromatic cells forming atypical glands. In other areas the tumor extended longitudinally between layers of the duodenal wall and outwardly

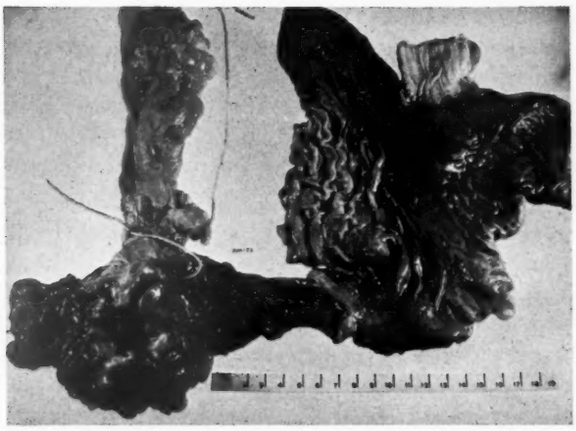


FIG. 1. Case 1. Autopsy specimen showing a large fungating carcinoma of the duodenum distal to the ampulla of Vater.

to serosa and mesentery. (Fig. 2.) Pathologic diagnosis was adenocarcinoma of the duodenum.

CASE II. A thirty-three year old white man was admitted to the Portland Veterans Administration Hospital on February 27, 1956, with a two-week history of nausea and vomiting of increasing severity. The vomiting was projectile in nature, occurring one to two hours after the ingestion of either solids or liquids. Associated with the present illness was progressive weakness, a 20 pound weight loss, constipation and mild abdominal cramps, but no hematemesis or melena. The past history was non-contributory except for an appendectomy in 1944.

Physical examination revealed a pale, thin, chronically ill man with significant findings limited to the abdomen. A healed scar was present in the right lower quadrant. Moderate distention was evident, but no organs or masses were palpable and tenderness was not elicited. The bowel sounds were hyperactive and rectal examination was negative.

Laboratory data were as follows: The red blood cell count was 4.76 million per cu. mm.; the white blood cell count was 11,400 per cu. mm., with a normal differential; urinalysis revealed a specific gravity of 1.014, albumin 2+, hyaline and granular casts. Serum electrolytes were: Na, 140 mEq./L.; K, 3.5 mEq./L.; Cl, 91 mEq./L.; and CO_2, 38 mEq./L. An upper gastrointestinal series done by a private physician just prior to the patient's admission to our hospital revealed obstruction in the upper jejunum with marked dilatation of the proximal segment. (Fig. 3.)

The patient was prepared for surgery by tube decompression of the obstructed jejunum and the restoration of fluid-electrolyte balance. On February 28th the patient was taken to surgery where a firm, pale yellow tumor of the jejunum was found 40 cm. distal to the ligament of Treitz. (Fig. 4.)

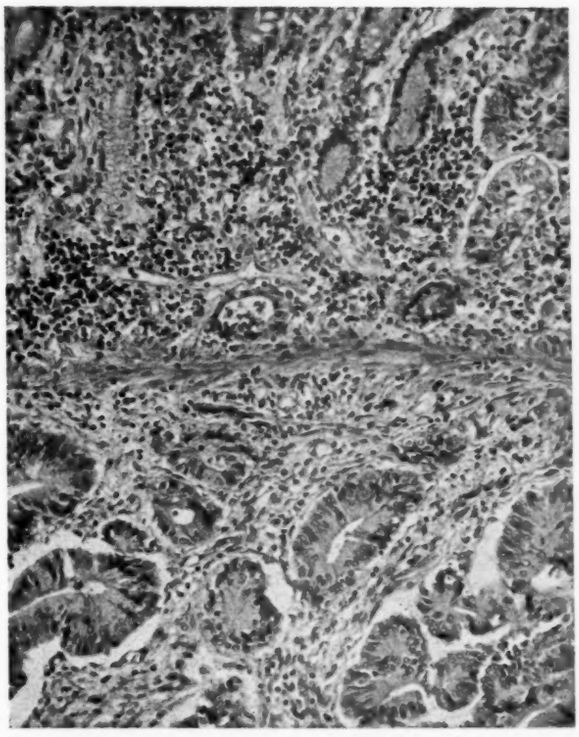


FIG. 2. Case 1. Low power magnification of the tumor showing infiltration of the wall of the duodenum.

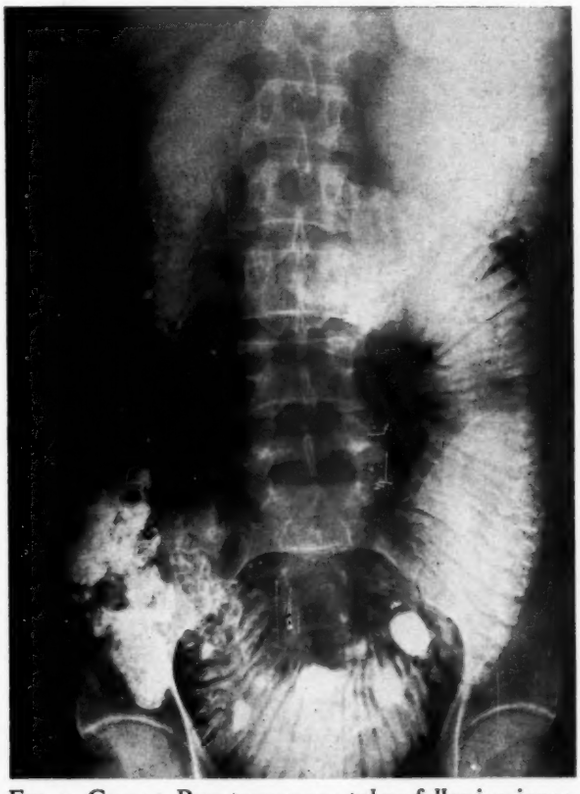


FIG. 3. Case II. Roentgenogram taken following ingestion of barium prior to admission to our hospital. The markedly dilated jejunum is demonstrated.

FIG. 4. Case 11. Photograph taken at surgery showing stenotic lesion of jejunum with serosal implants and enlarged mesenteric lymph nodes. Note the dilated proximal jejunum.

The bowel proximal to the lesion was dilated and its wall hypertrophied; distally the bowel was collapsed. The tumor completely encircled the jejunum and produced small bead-like serosal implants. Several mesenteric lymph nodes in close proximity to the main intestinal branches of the superior mesenteric artery lay adjacent to the lesion and were thought to be involved by metastatic tumor. The gross appearance of the lesion together with the age of the patient led to a diagnosis of lymphoma and no frozen section was performed. Because of the intimate relationship between the grossly involved lymph nodes and the superior mesenteric artery and the known response of lymphomas to radiation, a limited resection of jejunum and mesentery was performed, followed by an end-to-end anastomosis. The postoperative course was uneventful.

On pathologic examination the surgical specimen consisted of a 10 cm. segment of jejunum and attached mesentery. On opening the bowel specimen a fungating, pale yellow mass 3.5 cm. in diameter was found encircling the lumen and directly invading the mesentery. Three of five lymph nodes found in the mesentery were enlarged, firm and pale yellow on cut surface, the largest measuring 2 by 2.5 by 4.5 cm. in diameter.

Microscopically, sections through the margin of the tumor revealed an abrupt transition from normal mucosa to highly neoplastic tissue composed of irregular gland-like structures, the cells of which showed loss of mucus-secreting ability. Mitotic figures were numerous. (Fig. 5.) Sections of involved lymph nodes revealed complete replacement of normal architecture by nests of cells demonstrating definite gland formation. Pathologic diagnosis was adenocarcinoma of the jejunum with metastases to regional lymph nodes.

When the true nature of this tumor became known, it was believed that a more radical procedure should be attempted. Therefore on March 28th, the patient was returned to surgery where an extensive resection of the jejunum and mesentery, including the site of the previous anastomosis, was performed. The bowel continuity was re-established by an end-to-end anastomosis. The postoperative course was uneventful.

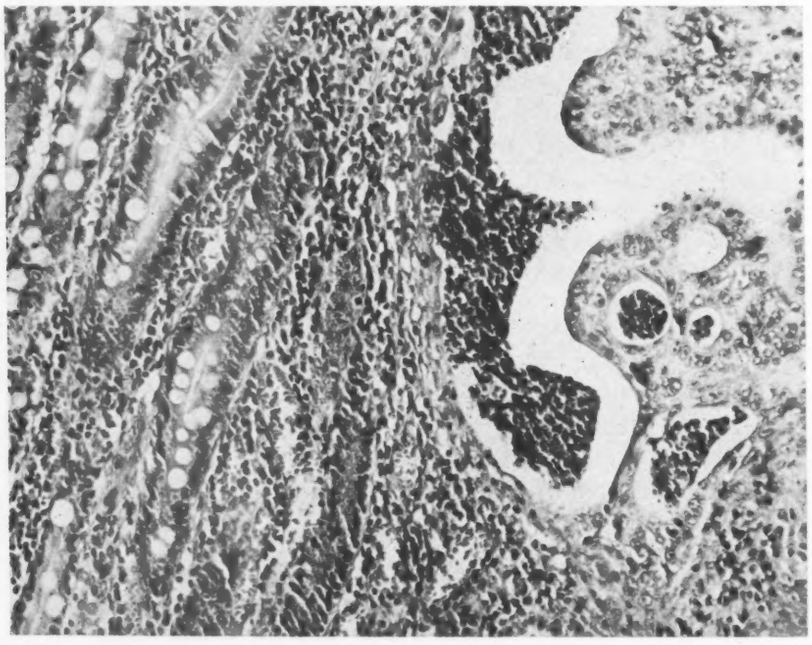


FIG. 5. Case 11. Low power magnification of adenocarcinoma of the jejunum with transition between normal mucosa (left) and tumor.

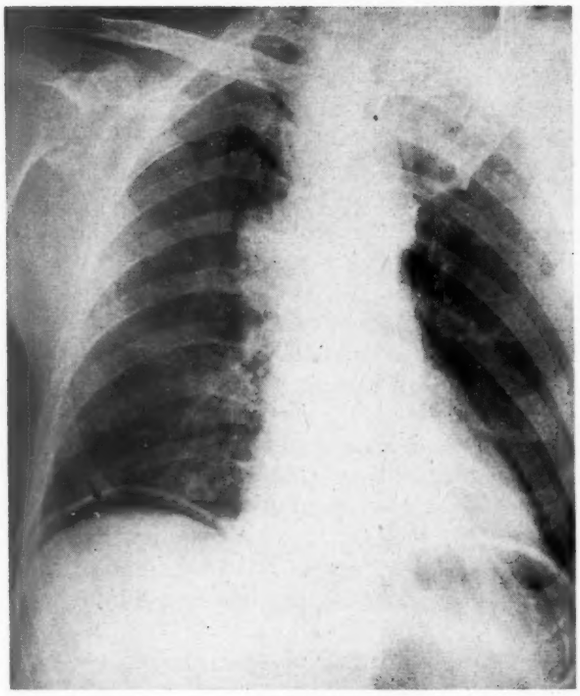


FIG. 6. Case III. X-ray film of the chest demonstrating air beneath the right leaf of the diaphragm.

The second surgical specimen consisted of 90 cm. of jejunum and attached mesentery. Both gross and microscopic examination of the bowel at the site of initial surgery and twenty-eight mesenteric lymph nodes failed to reveal any evidence of persistent tumor.

Since the patient's discharge from the hospital in April, 1956, he has maintained his preoperative weight and has returned to his job as an instrument repair man. At a Tumor Board recall on February 10, 1958, the patient had a normal red blood cell count; upper gastrointestinal and small bowel x-ray films showed no abnormalities, and he was asymptomatic in reference to the intestinal tract.

CASE III. A sixty-four year old white man, aphasic and hemiplegic secondary to a previous cerebrovascular accident, was admitted to the Portland Veterans Administration Hospital on March 22, 1957; his history was obtained from a nursing home supervisor. The patient had been in good health until two days prior to admission when he appeared ill and refused food but exhibited no nausea or vomiting. His condition deteriorated and on the day of admission he vomited dark material on several occasions. Bowel movements, regulated by periodic enemas, had been normal in color and there had been no jaundice or diarrhea. The past history was significant in that the patient had complained of abdominal distress partially relieved by amphojel® during a six-month period preceding the present illness.

FIG. 7. Case III. Surgical specimen showing perforated adenocarcinoma of the ileum.

Physical examination revealed an acutely ill, dehydrated, aphasic white man in obvious distress from abdominal pain. His abdomen was distended and tender to palpation, but no organs or masses were found. Bowel sounds were hyperactive and rectal examination revealed no abnormalities. The patient exhibited neurologic signs of right hemiplegia and motor aphasia.

Laboratory studies revealed the following: The red blood cell count was 4.21 million per cu. mm.; the white blood cell count was 23,800, with 81 per cent segmented neutrophils and 14 per cent staff cells. The blood urea nitrogen was 30 mg. per cent, while the serum electrolytes were: Na, 124 mEq./L.; K, 5.29, mEq./L.; Cl, 98.7 mEq./L.; and CO_2, 23 mEq./L. Radiographic examination revealed the presence of free air beneath the diaphragm. (Fig. 6.)

The admission diagnosis was perforated viscus with peritonitis and the patient was prepared for immediate operation by nasogastric suction, intravenous fluids and antibiotics. The patient was taken to surgery on March 22nd, at which time the most notable changes were found in the terminal ileum which was thickened and edematous with a perforation evident on the antimesenteric border 20 cm. proximal to the ileocecal valve. Adjacent to this area were numerous enlarged mesenteric lymph nodes. A limited segmental resection of the involved ileum was performed because of the poor condition of the patient; bowel continuity was re-established by end-to-end anastomosis.

On pathologic examination the surgical specimen consisted of a segment of ileum 21 cm. in length

FIG. 8. Case III. Low power magnification of adenocarcinoma of ileum with transition between normal tissue (left) and tumor.

with a small portion of attached mesentery. The bowel wall in the mid-portion of the specimen was thickened, firm and inflexible for approximately 7 cm. This area was covered by a thick fibrinous exudate and contained a perforation 1.5 cm. in diameter on the antimesenteric border. (Fig. 7.) On cut surface the mucosa and bowel wall were replaced by firm white tissue.

Microscopically, sections taken through the site of perforation revealed replacement of normal structures by neoplastic tissue composed of solid masses of cells with pleomorphic and hyperchromatic nuclei; mitotic figures were numerous. (Fig. 8.) Although no definite gland formation was seen, a periodic acid-Schiff (PAS) stain revealed pink-staining cytoplasm in the cells, indicating the presence of mucus. This established the diagnosis of primary undifferentiated adenocarcinoma of the ileum.

The patient's postoperative course was stormy, with high fever and slow return of intestinal peristalsis and appetite. Through prolonged parenteral fluid replacement and antibiotic therapy he regained his preoperative status and was discharged to a nursing home on April 9th.

The patient exhibited a progressive deterioration in his general condition and died on May 14, 1957. No autopsy was performed.

COMMENTS

Carcinomas of the small intestine are outnumbered 40 to 1 and 100 to 1 by malignant tumors of the colon and stomach, respectively [*14*]. Authorities disagree on the principle site of origin of these lesions, but nearly all writers agree that the proximal portion (duodenum and jejunum) is most frequently involved [*12,16*].

Clinically, cases of cancer of the small bowel may be classified into three groups on the basis of symptoms: bleeding or anemic, obstructive and perforative [*13*]. The majority of patients, however, have a combination of findings especially in respect to obstruction and anemia. Patients in the first group present findings of chronic blood loss: low hemoglobin, melena, weakness and occasionally hematemesis (Case I). It is extremely rare for massive hemorrhage to occur.

Individuals with an obstructing lesion give a history of vague, recurrent, cramping abdominal pain. The duration of symptoms varies with the location of the lesion, the average being nine months for carcinoma of the jejunum and six months for lesions of the ileum in one series [*15*]. Less frequently the onset is more acute [*8*] (Case II). The pain is usually aggravated by ingestion of food including bland foods, milk and antacids, an important point in differential diagnosis. Nausea and vomiting occur as the obstruction becomes more complete.

The perforative group is much less common, representing only 15 per cent of cases seen at Mayo Clinic [*15*]. However the patient presents a more dramatic picture with the sudden onset of abdominal pain associated with muscle rigidity, abdominal distention and roentgenographic evidence of free intraperitoneal air. A more detailed questioning of the patient will usually elicit a history of abdominal cramps, bloating or change in bowel habits present for several weeks or months prior to the actual perforation. The true diagnosis is usually made at the time of surgery. (Case III.)

Physical Findings. As a rule little is found on physical examination. The patient may appear pale and chronically ill; abdominal tenderness is common and frequently the only complaint. A palpable mass is present in 29 to 40 per cent of cases and is considered a late manifestation of the disease [*2,14*]. Patients

with acute obstruction or perforation present easily recognized abdominal findings and little time is lost in definitive treatment.

Age and Sex Incidence. Carcinomas of the small intestine behave in a manner similar to malignant tumors of the stomach and colon. They occur more frequently in males than females (approximately 2 to 1), and although found in patients in all decades are most common in those between the ages of forty and sixty years [*14*].

Roentgenographic Examination. The radiologist has assumed an important role in the diagnosis of tumors of the small bowel. An excellent review of the evolution of radiologic technics used in this field was made by Friedman and Rigler [*6*] in 1950. The following methods are utilized: flat plate of the abdomen; conventional barium swallow followed by interval x-rays as the barium progresses through the bowel; small bowel enema or the instillation of a thin barium solution through a long intestinal tube at various levels; and double contrast x-rays consisting of injection of barium followed by air into a "closed loop" of intestine created by use of a three-lumen tube. Finally, the terminal ileum can be visualized in a retrograde manner during a barium enema in patients with an incompetent ileocecal valve. Diagnostic patterns emphasized in the literature include obliteration of mucosal pattern and filling defects; suggestive findings are coiling and distention of the bowel, fixation of bowel wall, pseudodiverticula produced by hyperperistalsis, and delay in passage of barium [*5,7*]. Using these criteria certain authors have achieved 90 per cent accuracy in the preoperative diagnosis of tumors of the small bowel [*14,16*].

Diagnosis. Perhaps the most important factor in the diagnosis of tumors of the small bowel is the continued awareness of the existence of these lesions. Although the complaints in patients with cancer of the small intestine are frequently non-specific, a certain sequence of events should arouse suspicion. A patient with a diagnosis of peptic ulcer, chronic cholecystitis, chronic pancreatitis or psychoneurosis who does not respond to adequate medical management but continues to have abdominal pain, weakness, weight loss, melena and anemia or any combination of these findings should be considered to have a tumor of the small intestine [*4*]. This is especially true if no disease can be demonstrated in the stomach or colon. Certain authors place no value on the presence of occult blood in the stool [*3*]. As in many diseases, no one examination is diagnostic, but when combined with other findings it becomes a helpful adjunct in determining the nature of the underlying illness [*9,18*].

Treatment. The treatment is surgical excision. With the availability of new operative technics and advances in fluid-electrolyte therapy, the resectability rate has risen from 45 to 88 per cent in various series [*14,19*]. Jejunostomy, once recommended as a palliative procedure, was associated with a 75 per cent mortality [*8*]. Superior palliative methods for inoperable patients are gastroenterostomy or enteroenterostomy. However, wide resection of the involved bowel with its attached mesentery and lymph node–bearing tissue should always be performed when possible. The operative mortality at the Mayo Clinic dropped to an over-all rate of 22 per cent prior to 1947, with no deaths reported since 1935 [*15*].

Prognosis. There are numerous factors influencing the survival of patients with carcinoma of the small intestine. Some studies show an inverse relationship between grade of malignancy and five-year survival rates, those patients with grade III–IV lesions having a subsequent lower survival rate [*14,16*]. Locke [*10*], on the basis of his findings, found no such relationship and recommends wide resection in all cases. In general, the five-year survival rate for patients with cancer of the small bowel remains low, with delay in diagnosis being a major factor. In the last three decades, however, a rise from nearly zero to 12 per cent in 1940 and more recently to 66 per cent for operable lesions of the duodenum and 24 per cent for jejunoileal carcinoma has been noted [*11,20*]. As a rule cancers of the small bowel grow slowly, metastasize first to the regional lymph nodes, and only later to other organs such as liver, lungs and bone. With the continued development of new diagnostic technics and surgical procedures, the survival rate for patients with this disease will eventually reach a more satisfactory figure.

SUMMARY

1. Three cases of adenocarcinoma of the small intestine have been observed at the Portland Veterans Administration Hospital

since 1934, representing an incidence of 0.35 per cent of gastrointestinal malignancy.

2. Cases of cancer of the small bowel may be divided into three groups: anemic, obstructive and perforative. An example of each group was found in our series.

3. Any patient who does not respond to a specific program of treatment for abdominal disease or who shows persistent occult blood in the stool and anemia with no demonstrable disease in the stomach or colon should be thoroughly studied for carcinoma of the small intestine.

4. A preoperative diagnosis of tumor of the small bowel can be made in up to 90 per cent of cases when detailed radiographic examinations are undertaken.

5. The treatment is wide surgical excision of the involved bowel, mesentery and regional lymph nodes. In patients with distant metastases a more limited resection is justified.

REFERENCES

1. Anderson, W. A. D. Pathology. St. Louis, 1953. C. V. Mosby Co.
2. Bowman, P. G. Primary carcinoma of the jejunum and ileum. *Ann. Int. Med.*, 20: 779–788, 1944.
3. Cameron, A. L. Primary malignancy of the jejunum and ileum. *Ann. Surg.*, 108: 203–220, 1938.
4. Cattell, R. B. and Colcock, B. P. Tumors of the small intestine. *S. Clin. North America*, 27: 644–650, 1947.
5. Fraser, K. Malignant tumors of the small intestine. *Brit. J. Surg.*, 32: 479–491, 1945.
6. Friedman, J. and Rigler, L. G. A method of double-contrast roentgen examination of the small intestine. *Radiology*, 54: 365–379, 1950.
7. Gillette, P. C. Malignant tumors of the small intestine. *Gastroenterology*, 30: 517–522, 1956.
8. Heggs, F. M. Carcinoma of the jejunum: acute intestinal obstruction. *Brit. J. Surg.*, 26: 532–539, 1939.
9. Ingram, I. N. and Davis, W. C. Primary carcinoma of the jejunum. *Am. J. Surg.*, 87: 747–753, 1954.
10. Locke, D. J. Malignant neoplasms of the small intestine. *Am. J. Surg.*, 85: 127–133, 1953.
11. Mayo, C. W. Malignancy of the small intestine. *West. J. Surg.*, 48: 403–407, 1940.
12. Mayo, C. W. and Nettrour, W. S. Carcinoma of the jejunum. *Surg., Gynec. & Obst.*, 65: 303–309, 1937.
13. McComb, A. R. and Pridgen, J. E. Malignancies of the small intestine. *Ann. Surg.*, 145: 665–674, 1957.
14. Pollock, L. H. Tumors of the small intestine. *Am. J. Surg.*, 87: 851–859, 1954.
15. Pridgen, J. E., Mayo, C. W. and Dockerty, M. B. Carcinoma of the jejunum and ileum, exclusive of carcinoid tumors. *Surg., Gynec. & Obst.*, 90: 513–524, 1950.
16. Rabinovitch, J., Grayzel, D. M. and Swyer, A. J. Primary carcinoma of the small intestine. *Am. J. Surg.*, 80: 69–72, 1950.
17. Raiford, T. S. Tumors of the small intestine. *Arch. Surg.*, 25: 122–177, 321–355, 1932.
18. Rankin, F. W. and Mayo, C. Carcinoma of the small bowel. *Surg., Gynec. & Obst.*, 50: 939–947, 1930.
19. Strohl, E. L., Diffenbaugh, W. G. and Sarver, F. E. The surgical importance of small bowel tumors. *Surg., Gynec. & Obst.*, 93: 209–214, 1951.
20. Waugh, J. M. and Giberson, R. G. Radical resection of the head of the pancreas and duodenum for malignant lesions. *S. Clin. North America*, 37: 965–979, 1957.

Atypical Symptom-Sign Complex of Acute Pancreatitis

J. L. DONHAUSER, M.D. AND NOLTON H. BIGELOW, M.D., *Albany, New York*

From the Departments of Surgery and Pathology, Albany Medical College, and Albany Hospital, Albany, New York.

WHEN one considers the supposed cardinal symptoms and signs of massive acute pancreatitis, abdominal pain, vomiting and shock of varying degree are looked upon as typical of this disease. Indeed, the clinical picture which is usually described for fulminating extensive pancreatitis seems to present such a precise pattern that only moderate difficulty should ever be encountered in establishing a diagnosis. Nevertheless, the customary analysis of cases presented by most authors is usually replete with instances wherein the diagnosis was established quite unexpectedly, either at operation or autopsy. Consequently, it would appear that either the classic clinical profile of this disorder is deficient or that there is a deficiency of clinical awareness by the physician, or a combination of both factors.

The catastrophic or ultra severe type, so ably described by Dieulafoy [*13*] as the "pancreatic drama," necessarily enters our mind as being quite typical of the disease, yet from a clinical review of cases with necropsy findings, we find that some severe instances causing death within a few hours or a few days show little of the picture depicted by Dieulafoy.

An analysis of the clinical records of twenty-one cases verified by autopsy was therefore undertaken to ascertain how often an accurate diagnosis was made or at least how often it reasonably could have been made. While pancreatitis, either acute or chronic, is not an infrequent postmortem discovery, instances of massive acute pancreatitis of sufficient severity to be considered the major cause of death were relatively rare, only twenty-one cases being present in some 10,000 autopsies.

In evaluating the usually accepted clinical signs and symptoms, an attempt has been made to show that in many cases the classical signs and symptoms were not present.

Pain, generally of a severe degree, is considered as one of the outstanding features of acute pancreatitis. (Table 1.) Various characteristic features of this pain together with other signs and symptoms are usually considered sufficiently unique as to be virtually pathognomonic of this disorder. Furthermore, the magnitude of the pathologic process is believed to be directly proportional to the severity of the pain. The analysis and description of the pain in pancreatitis are so uniform in discussion

TABLE 1
ACUTE HEMORRHAGIC PANCREATITIS

No. of Cases	Site of Pain	Clinical Diagnosis
4	Epigastrium	(1) Common duct stone, possible appendicitis, gastric ulcer, intestinal obstruction; (2) intestinal obstruction, gastric ulcer; (3) coronary occlusion; (4) acute gastric indigestion
3	Right upper quadrant	(1) Common duct stone; (2) intestinal obstruction; (3) intestinal obstruction
1	Left upper quadrant	Splenic infarct or pulmonary embolism
6	Generalized abdominal	(1) Intestinal obstruction; (2) probable acute pancreatitis; (3) perforated gastric ulcer; (4) coronary occlusion or gallbladder disease; (5) intestinal obstruction, adynamic ileus cirrhosis, alcoholism; (6) probable cholecystitis with calculi, pancreatitis, possible tuberculous or carcinomatous peritonitis
1	Precordial	Acute coronary insufficiency or questionable pancreatitis
4	No pain	(1) Kidney shutdown; (2) homologous serum disease; (3) coronary occlusion, hypertensive heart disease; (4) arteriosclerotic heart disease
1	Shock	Delerium tremens, chronic alcoholism
1	Incoherent	Hypertensive heart disease, syphilis of heart

given by a multitude of authors that little if any possibility of misunderstanding should seem justifiable. Indeed, in a recent article written by Donhauser [4], reviewing eighty-six cases it was stated: "Acute pain, especially referred to the epigastrium and right upper quadrant, was present, although in the most severe cases, acute diffuse abdominal pain was the first symptom." A review of the autopsy records correlated with the clinical charts, however, shows that in the most severe cases patients may present with no abdominal pain at all. Despite the clear clinical picture that emerges with almost textbook clarity it is well known that this disorder is difficult to establish clinically. Perhaps inadequate evaluation and understanding of the nature of pain in pancreatitis are important factors in diagnostic error. Other possible sources of error from the clinical or laboratory standpoint will only be considered herein insofar as they pertain to and correlate with the phenomenon of pain in pancreatitis. The character and severity of pain in acute pancreatitis are considered virtually diagnostic. Great emphasis is placed on the epigastric nature and its excruciating severity. Such terms as "most painful of abdominal catastrophes" are frequently applied. However, there are cases in which the pain is atypical as to its location and of only moderate severity and in which at times there is complete absence of pain. As our cases show, some patients were in shock with virtual coma, perhaps too ill to complain of such pain, while in others with no sign of presenting shock, pain was entirely absent. However, the lack of excruciating pain in cases of fatal acute pancreatitis should not be too surprising for a similar situation exists regarding pain in myocardial infarction. Truly, the excruciating pain of myocardial infarction is almost beyond human endurance. Yet it is too well known to require further elaboration that a severe and even mortal myocardial infarct may be virtually symptomless. The absence of pain would be considered a most inadequate reason for not obtaining an electrocardiogram.

As our analysis proceeds, it will be shown that while other signs and symptoms are variable but little attention has been paid to the possibility of acute pancreatitis being heralded by a mere "touch of indigestion," slight epigastric fullness, or if there is pain, that it is related to possible gallbladder disease, peptic ulcer or other origin. Consequently, no amylase determination is made. An inescapable conclusion derives from the marked variability of the pain pattern; therefore, in any kind of upper abdominal disorder of uncertain or obscure nature, an amylase determination should be made. Bartholomew [2], in a recent report of ten cases of chronic pancreatitis stated that chronic pancreatitis may progress to extensive destruction of the pancreas in the complete absence of pain.

The sites of acute pain in our series were as follows: epigastrium, four; right upper abdominal quadrant, three; left upper abdominal quadrant, one; generalized abdominal, six; precordial, one; and no pain, four. One patient was in shock and one in coma.

That so little consideration was given to the possibility of the existence of acute pancreatitis is demonstrated by the fact that in only six cases was the amylase determination made.

Too much emphasis, therefore, should not be placed upon the site or even the absence of pain in this condition. We are, of course, aware that acute abdominal pain is of the greatest symptomatic importance, but that the disease may very definitely exist with a most bizarre type of pain or even the absence of same.

An analysis of these six cases fails to disclose the reasons for a tentative diagnosis of acute pancreatitis not having been made, with the exception of Case III in which the patient exhibited the sign-symptom complex of this disease. In seven cases the symptoms and signs were so typical of cardiac disease that the taking of an amylase evaluation was not even considered. Varied diagnoses were made, such as coronary occlusion, cardiac insufficiency, intestinal obstruction, arteriosclerotic disease, chronic alcoholism, biliary tract disease, splenic infarct and perforated gastric ulcer. The records of three cases show that despite marked elevation of the serum amylase levels, other diagnoses were made.

We know that a rise in amylase may occur in varied diseases although it is our belief that when a rise does occur, the first consideration should be made as to the probability of the existence of acute pancreatitis.

Cyanosis and dyspnea were present in twelve cases and it is surprising that on the average, of eighteen cases noted, the respirations averaged only 24.

Temperature is variable and is usually not

high unless accompanied by extensive necrosis and abscess formation.

Pulse is considered as of diagnostic importance and yet in this review, of the eighteen pulses which could be taken accurately, the average count was 100 and of good quality. Three pulses were imperceptible.

Table II
ACUTE HEMORRHAGIC PANCREATITIS
Correlation between Blood Sugar and Urinary Sugar

Urinary Sugar	Blood Sugar	Acetone
Not taken	586	2+
Trace	320	...
Trace	144	+
Trace	220	...
Trace	105	...
None	180	...
None	100	...
None	165.3	+
4+	451	...
Trace	119	...
1+	296	+
None	140	...
2+	304	...
1+	225	...

Shock was present in only three of the twenty-one cases. In the remaining patients, no evidence of shock was observed. The average systolic pressure for the entire series was 128 mm. Hg. and the average diastolic pressure 78 mm. Hg.

Vomiting, often excessive and continuous, occupies a most prominent place in a symptom complex of this disease. In this series, vomiting was present in only ten patients.

Abdominal tenderness is so commonly found, either in the upper quadrants of the abdomen or generalized, that this sign must be considered as an aid in diagnosis. This sign was noted in fifteen of our patients; there was no tenderness in one, and of five, no mention is made.

"Rigidity is usually present, but as a rule, not as marked as in other acute abdominal conditions, such as perforated ulcer and the like. However, in our experience, rigidity is of great diagnostic importance, tho' in cases in 'Extremis,' it may not be present" (Donhauser). Christian [3] states "There may be a distinct Extremis, it may not be present" [4]. Christian [3] mentions that "There may be a distinct sense of resistance but marked rigidity is usually absent." Homans [8] concludes that "Muscular resistance may obscure the diagnosis."

In our series, rigidity was present in seven patients, the abdominal muscles were noted as being tense in two, and rigidity was absent in four. No mention of rigidity was made in the other eight patients.

Table III
ACUTE HEMORRHAGIC PANCREATITIS
Positive Electrocardiogram (Ten Cases)

Age (yr.) and Sex	Duration	Clinical Diagnosis
61, M	5 wk.	Coronary occlusion
23, F	7 days	Perforated gastric ulcer
58, M	8 hr.	Coronary occlusion
56, F	36 hr.	Splenic infarct, pulmonary embolism
65, F	3 wk.	Homologous serum disease
54, M	4 days	Intestinal obstruction
67, F	6 hr.	Common duct calculus, intestinal obstruction
63, F	24 hr.	Acute coronary insufficiency, questionable pancreatitis
33, F	6 days	Sinus tachycardia, nephrosis
83, M	2 wk.	Coronary occlusion

Obesity is a recognized portion of a symptom-sign complex in this disease, but it was only noted in the records as having been present in ten patients. It is our belief, however, that obesity was present in other cases although not specifically mentioned in the records.

Four patients were definitely addicted to alcohol.

The use of the laboratory cannot be over emphasized. The presence of glycosuria, together with hyperglycemia, is certainly of diagnostic importance, but the non-correlation of the two is of such significance that extreme moment should be considered in the presence of hyperglycemia without an accompanying glycosuria. (Table II.) It should be noted that eight patients showed glycosuria while eight showed an average of 240 mg. blood sugar. White blood counts in the fifteen patients recorded showed an average of 14,504 per cu. mm. Wassermann reaction was noted in one case.

Relation to blood vessel disease was as follows: (Tables III and IV). Electrocardiogram was noted as abnormal in ten cases and questionable in one. Eight patients were clinically diagnosed as affections of the heart. Popper et al. [10], have brought out the fact that experimentally, temporary occlusion of the main pancreatic (gastro-

TABLE IV
CLINICALLY DIAGNOSED AS HEART AFFECTIONS

Age (yr.) and Sex	Chief Complaint	Duration	Pulse	Respiration	Blood Pressure (mg. Hg)	Amylase	Electrocardiogram	Clinical Diagnosis
62, M	Dyspnea	1 yr.	88	22	140/70	Not taken	..	Arterial sclerotic heart disease
61, M	Dyspnea	5 wk.	84	36	100/40	Not taken	+	Coronary occlusion
63, F	Precordial pain and pain in left upper abdominal quadrant	24 hr.	80	18	140/80	Not taken	+	Coronary insufficiency, highly questionable pancreatitis
33, F	Kidney shutdown	6 days	130	30	.....	Not taken	+	Sinus tachycardia, nephrosis, diabetes
83, M	Epigastric pain	2 wk.	70	18	120/60	Not taken	+	Coronary occlusion
58, M	Abdominal pain	8 hr.	120	24	160/80	1,500 units	+	Coronary or gallbladder disease
52, M	Incoherent	.....	...	..	.....	Not taken	..	Hypertensive heart disease, syphilis of heart
58, F	Pain in right upper quadrant	4 wk.	134	..	150/78	Not taken	..	Cardiac decompensation

duodenal) artery performed in animals with edema of the pancreas led to the development of pancreatitis. Local vasospasm and ischemia may play an important role in the development of acute pancreatitis and it is observed that temporary local ischemia can transform clinically harmless transient edema of the pancreas into hemorrhagic necrosis. Garster et al. [7], point out the fact that acute hemorrhagic pancreatitis may occur not only through obstruction to the main pancreatic duct but also from a vascular abnormality within the parenchyma of the pancreas. Dunphy [5] mentioned the fact that in older people, arteriosclerosis will produce intermittent pancreatitis of a mild type but which is occasionally fatal. Baker [1] believes that obliteration of the blood supply may play a role and that at autopsy the pancreatic arteries deserve the "most careful attention." A factor of probable importance in many instances is ischemia. Experimentally, interference with the pancreaticoarterial supply has been shown to enhance the development of pancreatitis after ligation of the pancreatic duct, hormonal stimulation or both and there is clinical evidence that vascular lesions are significant etiologic factors. Deterioration in local blood supply may be a most important factor. The appended tables indicate how clinically this has often been mistaken for a circulatory affection.

TABLE V
ACUTE PANCREATITIS

Condition of gallbladder at operation or autopsy (absence or presence of calculi):	
Negative gallbladder with no calculi	11
Chronic cholecystitis with calculi	7
Chronic cholecystitis with no calculi	3
Relation of amylase findings with clinical diagnosis (only six amylase determinations taken):	
Common duct stone, possible appendicitis, gastric ulcer or intestinal obstruction	500
Splenic infarct or pulmonary embolism	152
Probable acute pancreatitis	452
Perforated gastric ulcer	4,368
Coronary occlusion or gallbladder disease	1,500
Homologous serum disease, obstructive jaundice	620

Menguy [9], in experimentally induced acute pancreatitis in dogs and cats, has ascertained that "When ligation of the ducts was combined with ligation of the major pancreatic arteries or veins or with stimulation of the left splanchnic nerve, acute pancreatitis of a severe degree ensued"; but he made the comment that "Vascular disturbances alone were not sufficient to induce acute pancreatitis."

No attempt has been made to discuss the varied theories of the causes of acute massive pancreatitis. A review of our cases with findings at autopsy and clinical notes have made us seriously consider the direct relationship be-

tween this disease and the vascular system, as noted by Baker and Boles [*1*]: "At autopsy, a careful dissection of the main and accessory ducts and relationship to the biliary tract, plus a search for stone, stricture metaplasia and dilatation and the state of the pancreatic arteries are details which deserve the most careful attention."

It is interesting to note that in a recent communication, Probstein et al. [*12*] reported twelve cases " . . . showing atheromatous embolization from the aorta to the pancreatic arteries, in 10% of which there was an associated acute pancreatitis." From these observations Probstein concluded that there may be a relationship between the parenchymatous reaction and the number of pancreatic vessels involved in the embolic process. In the study of our patients presented in this brief, it is to be regretted that a dissection of the pancreatic arteries had not been more definitive, especially in the age group in which disease of the blood vessels was so common.

We know that the pathologic picture in pancreatitis is one of edema which in severe cases terminates in necrosis and hemorrhage. That the necrosis is due to the action of a pancreatic enzyme is quite well established. Powers et al. [*11*], in a recent article, emphasize the fact that "The transition from pancreatic edema to necrosis, follows the conversion of trypsinogen to trypsin within the pancreas and it seems likely that serum trypsinogen levels are a highly sensitive and specific test for hemorrhagic pancreatitis."

It is well known that certain clinical tests, such as serum amylase, are most useful in establishing an accurate diagnosis if performed in the early acute stages of acute pancreatitis. However, normal values are often obtained after the disease has been present for two or three days. Since this laboratory test is so relatively simple the only reason for not performing it must lie in the fact that no one thinks of the possibility of pancreatitis early in the course of this disorder. Why this is so is not entirely clear, unless the classic patterns allegedly characteristic of this disorder are not encountered with sufficient frequency to excite suspicion as to the correct nature of the illness. For this reason analysis of the various facets of the clinical picture seems warranted. (Table v.)

CONCLUSIONS

Accurate diagnosis in many cases of acute pancreatitis is not made due to the absence of the so called symptom-sign complex of the disease.

Pain may be entirely absent or it may be so insignificant in nature as to mitigate (militate) the thought of the existence of pancreatitis.

The absence of pain is analogous to the severe and ever mortal occurrence of a myocardial infarct.

Cardiovascular disease may be confused with pancreatitis as has been noted in this series.

There may be a direct relation between the pathologic condition of the cardiovascular system and this affection.

There is a non-correlation between glycosuria and hyperglycemia and these findings should always be considered as of possible diagnostic importance.

In any type of upper abdominal disorder of uncertain or obscure nature, an amylase determination should be made.

REFERENCES

1. Baker, J. W. and Boles, T. Observations pertaining to the place of surgery in acute pancreatitis. *Gastroenterology*, 28: 536–549, 1955.
2. Bartholomew, L. G. and Comfort, M. W. Chronic pancreatitis without pain. *Proc. Staff Meet., Mayo Clin.*, 14: 361–364, 1957.
3. Christian, H. A. Principles and Practice of Medicine, p. 776. New York and London, 1947. Appleton-Century-Crofts.
4. Donhauser, J. L. Acute pancreatitis. *Am. J. Surg.*, 88: 946–952, 1954.
5. Dunphy, J. E. The pathogenesis of acute and chronic pancreatitis (in discussion). *Ann. Surg.*, 142: 690–697, 1953.
6. Elman, R. Acute pancreatitis. *J. Nat. M. A.*, 386–397, 1954.
7. Garster, J., et al. The pathogenesis of acute pancreatitis. *Arch. Surg.*, 60: 473–491, 1950.
8. Homans, J. A Text Book of Surgery, p. 893. Springfield, Illinois, 1940. Charles C Thomas.
9. Menguy, R. B. et al. Ductal and vascular factors in the etiology of experimentally induced acute pancreatitis. *Arch. Surg.*, 74: 881–889, 1957.
10. Popper, H. L. et al. Transition of pancreatic edema with partial necrosis. *Surg., Gynec. & Obst.*, 87: 79–82, 1948.
11. Powers, S. H. et al. The pathogenesis of acute and chronic pancreatitis. *Ann. Surg.*, 142: 670–697, 1955.
12. Probstein, J. B. et al. Atheromatous embolization, an etiology of acute pancreatitis. *Arch. Surg.*, 4: 566–572, 1957.
13. McCrae, T. Medical aspects of acute pancreatitis. *Atlantic M. J.*, 28: 555–558, 1925.

Acute Emphysematous Cholecystitis

A CASE REPORT AND REVIEW OF THE WORLD LITERATURE

ALFRED EDINBURGH, M.D. AND ABRAHAM GEFFEN, M.D., *New York, New York*

From the Department of Surgery and from the Department of Radiology, Beth Israel Hospital, New York, New York.

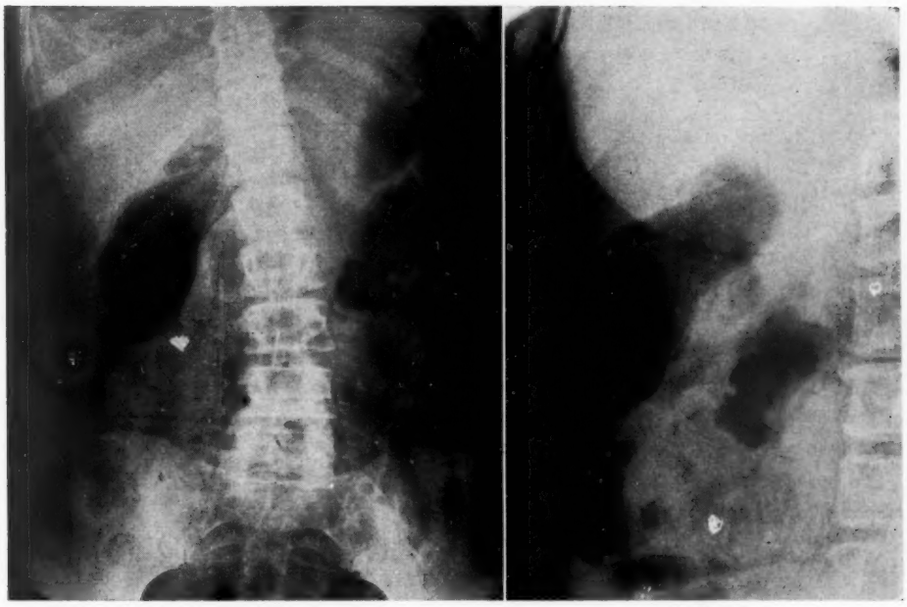
THIS is the fiftieth case reported in the medical literature of the clinical and pathological entity known variously as acute pneumocholecystitis, pyopneumocholecystitis, acute gaseous cholecystitis, gas gangrene of the gallbladder and emphysematous cholecystitis. The purpose of this paper is to document an additional case of acute emphysematous cholecystitis, review the cases previously reported in the medical literature, review and discuss the pathogenesis and clinical course of this condition and evaluate the methods of treatment, especially as demonstrated by the findings in this case.

CASE REPORT

Mrs. H. L., a sixty-two year old white woman, was admitted to Beth Israel Hospital for the first time on July 2, 1956, with a four- to five-day history of severe, intermittent abdominal pain in the right upper quadrant associated with interscapular radiations. She had been receiving antibiotic therapy at home for several days prior to admission to the hospital. Seven or eight years ago attacks of epigastric pain developed radiating to the interscapular area. Since onset these attacks have recurred intermittently, usually clearing within twenty-four hours.

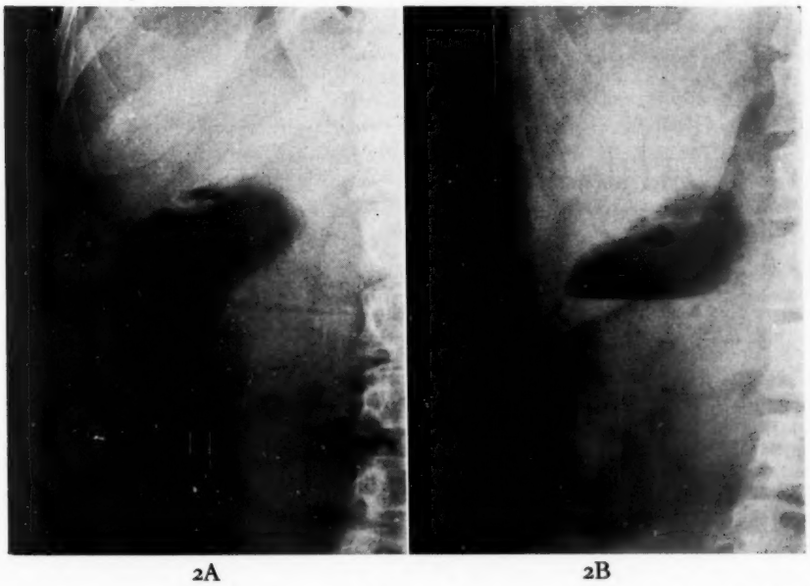
On admission the temperature was 101.8°F., pulse 84, blood pressure 132/84 mm. Hg. The patient was an obese, well hydrated white woman complaining of diffuse abdominal pains. The patient did not seem to be acutely ill at any time after admission to the hospital and appeared to be quite comfortable. There was limited diaphragmatic excursion on the right side of the chest with slight dullness and decreased breath sounds at the right base posteriorly. There was marked tenderness with spasm and guarding in the right upper quadrant of the abdomen with local and referred rebound especially from the right upper quadrant. Right costovertebral angle percussion elicited right anterior subcostal pain.

Laboratory examinations revealed brown, acid urine with specific gravity 1.020; no albumin, glucose or acetone, 5 to 6 white blood cells per high power field, loaded with epithelium and negative for bile; 11.8 gm. of hemoglobin, 14,600 white blood cells per cu. mm. with 72 per cent polymorphonuclear leukocytes, 8 per cent staff forms, 1 per cent lymphocytes and 14 per cent monocytes; and nine days later 16,550 white blood cells per cu. mm. The fasting blood sugar was 112 mg. per cent; bilirubin 2.47 mg. per cent on admission and 1.27 mg. per cent one week later. The cephalin flocculation test was negative; blood culture, while the patient was receiving penicillin and streptomycin, showed no growth. On admission, x-ray examination of the abdomen, including special views of the right upper quadrant, showed the gallbladder to be outlined with gas. (Fig. 1A and B.) The gallbladder, appeared to be distended with the gas in the lumen. There definitely was gas within the wall of the gallbladder. No gas was seen in the biliary ducts. There was also moderate distention of several loops of small bowel and minimal amounts of gas in the colon. The impression was acute emphysematous cholecystitis. The following day the x-ray examination of the abdomen showed the gallbladder to be distended and again completely filled with gas which extended into the wall. There still was segmental distention of small and large bowel. On July 9th, seven days after admission, the roentgenograms of the abdomen (Fig. 2A and B) showed the gallbladder to be still outlined with gas within the lumen and also within the wall. The film taken with the patient erect showed a fluid level within the gallbladder, and there appeared to be slightly less distention of the gallbladder. There was also noted mottling within the lumen, suggestive of radiolucent calculi. Finally, on July 17th, fifteen days after admission (Fig. 3A and B), films of the right upper quadrant, including an erect view, showed a definite decrease in the



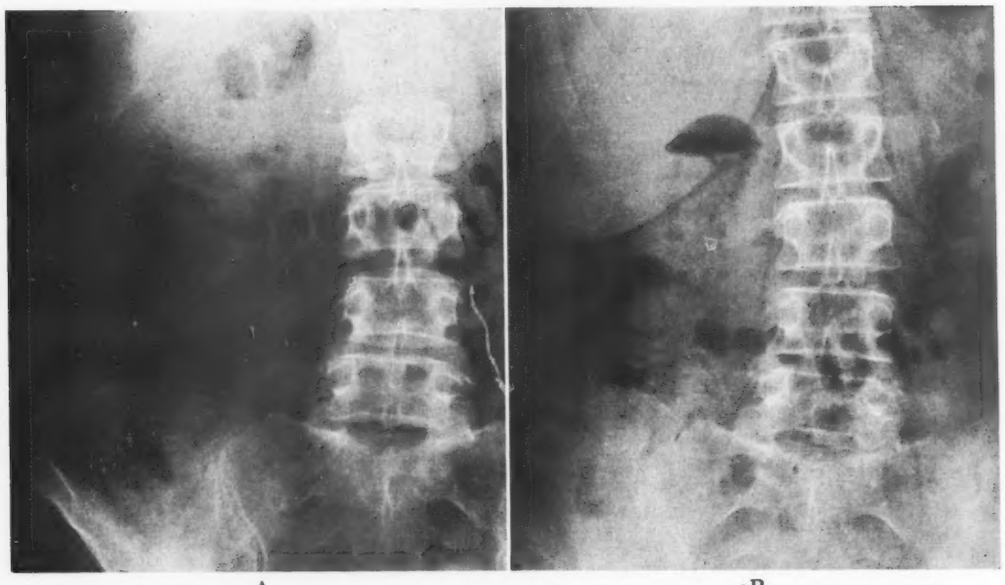
1A 1B

FIG. 1. A, film of the abdomen with the patient supine, on day of admission, shows the gallbladder distended with gas in the lumen. Gas is also seen within the wall of the gallbladder. No gas is seen in the biliary ducts. B, posterior-anterior view of the right upper quadrant on day of admission, shows distended gallbladder with gas in the lumen and within the wall.



2A 2B

FIG. 2. A, posterior-anterior view of the right upper quadrant taken six days after admission still shows gas in the lumen and within the wall of the gallbladder. The gallbladder is less distended. The mottling within the lumen suggests the presence of radiolucent calculi. B, erect view of the right upper quadrant taken six days after admission demonstrates the fluid level within the gallbladder, confirming the presence of intraluminal gas. The intramural gas is also demonstrated.



3A 3B

FIG. 3. A, prone view of the right upper quadrant taken fifteen days after admission shows only mottling in the region of the gallbladder, suggesting that the gas may have completely disappeared. B, erect view of the right upper quadrant taken fifteen days after admission shows definite fluid level, indicating that gas is still present in the lumen of the gallbladder. The amount of gaseous distention has decreased considerably. This view shows very faint evidence of intramural gas.

amount of air in the gallbladder. Air could still be demonstrated in the wall of the gallbladder, and an air-fluid level was again noted.

The patient's clinical course was notable in that at no time did she appear acutely ill. The diagnosis of acute emphysematous cholecystitis having been established on the day of admission by the x-ray examinations, the patient was treated accordingly. Immediate surgical intervention was not undertaken because of the clinical evidences that the process was under control and subsiding. She was placed on a regimen of terramycin® 200 mg., administered intramuscularly four times a day; procaine penicillin 1,000,000 units, administered intramuscularly three times a day; and aqueous penicillin 200,000 units, administered intramuscularly every two hours. Nothing was administered orally. Intravenous fluids were also given. The temperature ranged from 100 to 101°F. for one week. After two days on this regimen the patient improved enough to tolerate oral fluids and then a soft, fat-free diet. The clinical signs of an acute process gradually cleared and the gallbladder was less distended on x-ray examination after one week. After two weeks, because of an allergic reaction to penicillin, she was treated with chloromycetin® 500 mg. every six hours. At that time, although the x-rays as just described showed gas persisting in the lumen and wall of the gallbladder, there were no longer any local or generalized evidences of an acute cholecystitis. When the patient had remained afebrile for two days without the use of antibiotics, she was discharged home with instructions to return for an interval cholecystectomy.

She was readmitted August 6, 1956, about six weeks after the onset of the acute attack. She had been well in the interim and her abdominal examination revealed no abnormalities. Operation on August 8th was performed through a modified right subcostal incision. A large inflammatory mass was revealed occupying the right subhepatic space. The omentum and transverse colon were adherent to the liver edge. The gallbladder was not immediately visualized. It was felt that a subhepatic pericholecystic abscess was present. After thoroughly padding off the abdominal cavity and wound edges from the operative area, the omentum was separated from the liver in the vicinity of the gallbladder. An abscess containing a few ounces of pus was entered. The pus was thick, white, nonodorous, and showed no evidence of bile. When the abscess cavity had been evacuated, a structure resembling the gallbladder could be seen lying freely within it. This structure was picked up with a sponge forceps and gently lifted out of the cavity. This proved to be the gallbladder, which was completely free and contained numerous small stones. There were no stones in the pericholecystic abscess. The gallbladder apparently had not grossly perforated. A catheter was placed down to what appeared to be the cystic duct and sutured in place. A cigarette drain was also placed into the abscess cavity. Although no bile was noted

at the time of operation, bile drainage was noted on the following day through the catheter.

Postoperatively the patient was placed on a regimen of chloromycetin 0.5 gm. twice a day. Bile was draining via the tube drain and oral fluids were tolerated the first day. The temperature ranged from 100 to 100.8°F. for five days. The operative culture showed coliform organisms sensitive to chloromycetin and aureomycin.® The final bacteriology report was rare gram-negative rods, Bacterim coli, Bacterim lactis aerogenes and Bacillus pyocyaneus, but there was no evidence of clostridia. One drain was removed four days postoperatively, but purulent material continued to drain intermittently via the tube. Chloromycetin was discontinued after eight days when the patient had been afebrile for three days. Sutures were removed ten days after the operation, but bile continued via the drainage site until the twelfth day. Therefore, lipiodol was instilled through the tube fourteen days after surgery, revealing a narrow tract in the right upper quadrant of the abdomen corresponding to the drainage tube; there was no communication with the biliary tree. The tube was removed and the patient discharged after sixteen days.

Pathological examination showed a gallbladder measuring 11 cm. from the fundus to the neck and 2.5 cm. in diameter at the fundus. The external surface was roughened by fibrous strands. The wall of the gallbladder measured 0.1 cm. in thickness; the mucosa was bile-stained and reticulated. The lumen contained dark brown bile and numerous black, rough-surfaced friable concretions ranging from 0.1 cm. to 0.6 cm. in diameter. Microscopic examination reported necrotic autolyzed tissue suggestive of a portion of the wall of the gallbladder; no definite muscular layer was observed. In one area elements suggestive of the villous pattern of the gallbladder mucosa was present. Bacterial colonies were present.

COMMENTS

History. The condition of acute emphysematous cholecystitis was first described over fifty years ago by Stolz [*47*], in 1901, in three patients with gas in the gallbladder and ducts found at autopsy. Lobinger [*28*], in 1908, first described gas in the gallbladder lumen associated with gas within its walls found at operation for acute cholecystitis. The first preoperative diagnosis was made by Hegner [*15*], in 1931, on the basis of an x-ray film of the abdomen showing a pear-shaped gas shadow in a sixty-two year old man with the symptoms of acute cholecystitis.

It is interesting to note that of the fifty cases recorded in the medical literature, only seven were reported prior to 1940, but thirty-six were reported from 1945 to 1957. (Table 1.)

Bacteriology. Bacteriological studies revealed a wide range of organisms. Anaerobic cultures, despite their importance, were not taken in at least eleven instances. Clostridia welchii alone, or in combination with B. coli, was most frequently cultured. Clostridia were noted in about 40 per cent of cases, and B. coli in 30 per cent of cases in which cultures were taken. The various organisms cultured and their frequency of occurrence were as follows: B. coli, 11; Cl. welchii, 7; Staphylococcus aureus, 8; anaerobes (unidentified), 4; Clostridium perfringens, 3; streptococci, 3; Clostridium oedematiens, 1; Clostridium filaformi, 1; anaeraerobic Clostridium bellonansis, 1; Bacillus aerogenes capsulatus, 1; Streptococcus fecalis paracolon, 1; Aerobacter aerogenes, 1; and B. pyocyaneus, 1. In our patient, cultures which were taken at operation after subsidence of the acute phase revealed B. coli, B. lactis aerogenes, and B. pyocyaneus.

Studies by Gordon-Taylor [*12,13*] on the bacteriology of cholecystitis, exclusive of the emphysematous variety, have shown an incidence of non-sporulating Cl. welchii of low virulence of 18 per cent in operated gallbladders, and 13 per cent at autopsy. Cl. welchii were most frequently found in the gallbladder wall and in calculi, and less frequently in bile [*42*]. The liver and bile radicles are a normal habitat for the gram-positive anaerobic organisms. With injury in the biliary system or to the liver, or anoxemia in the wall of the gallbladder with necrosis of tissue (as occurs in acute cholecystitis) a medium for the growth of anaerobic organisms is established, and the next step is the development of gangrenous cholecystitis. Therefore, we may say that the growth of gas-forming organisms is probably secondary to, and not responsible for, the development of acute cholecystitis.

Clinical Picture. The clinical picture is usually that of an acute cholecystitis, but with a more abrupt onset, in most instances, of epigastric pain and/or pain in the right upper quadrant, sometimes with radiations. Sometimes the patient is acutely ill with a temperature elevation up to 103°F., but very often there is either no temperature elevation or a low grade fever ranging from 99 to 102°F. There are the usual signs of tenderness and

TABLE I

CASES OF EMPHYSEMATOUS CHOLECYSTITIS COMPILED FROM THE WORLD LITERATURE

Case	Author and Year	Age, Sex	Diabetes	Calculi	Diagnosis	Treatment and Result	Mass	Jaundice	Culture	Pathology
1	Lobinger, 1908	55, M	Yes	No note	Operation	Cholecystectomy 4 days after onset. Recovered.	No	No	Lost	
2	Tanner, 1921	44, M	No	None	Operation	Cholecystectomy 5 hrs. after onset. Recovered.	No	No	Streptococci and B.coli	Acute, gangrene, distended, air.
3	Kirchmayr, 1925	64, M	Yes	Present	Operation	Cholecystectomy 3 days after onset. Recovered.	No	No	Cl.welchii (non-sporing)	Distended, gas.
4	Hegner, 1931	62, M	No	Present	X-ray	Cholecystectomy 12 days after onset. Died.	Yes	Yes	Cl.welchii	Acute, distended, gas, necrosis, abscess
5	Simon, 1932	32, M	No	Present	X-ray	Cholecystectomy 6 days after onset. Recovered.	No	No	Anerobic sporules, bacilli	Acute, distended, gangrene, abscess.
6	Wybauw, 1931	53, F	No	Present	X-ray	Cholecystectomy. Recovered.	Yes	No		Distended, gas, abscess.
7	Schmidt, 1938	37, M	No	No note	X-ray	Conservative; bed rest. Recovered.	No	No	None	
8	DelCampo and Otero, 1940	74, M	No	Present	X-ray	Cholecystectomy. Died.	Yes	No	Cl.perfringens	Distended, gas, gangrene, abscess, peritonitis.
9	McCorkle and Fong, 1942	49, M	No	None	X-ray	Cholecystostomy 3 days after onset. Died.	No	No	Cl.welchii and B.coli	Died 2 days postoperatively with emphysema of abdomen, chest wall, and wound.
10	McCorkle and Fong, 1942	59, M	No	No note	X-ray	Conservative: sulfathiazole, polyvalent antisera. Recovered.	Yes	No	B.coli and clostridia	
11	McCorkle and Fong, 1942	49, M	No	No note	X-ray	Conservative: sulfathiazole, polyvalent antisera. Recovered.	Yes	No	B.coli, Cl.welchii and staphylococci	
12	Stevenson, 1940	64, M	Yes	Present	X-ray	Cholecystostomy 8 days after onset. Recovered.	Yes	Yes	Aerobic and anerobic streptococci	Distended, gas, gangrene, abscess.
13	Stevenson, 1940	63, M	No	Present	X-ray	Cholecystostomy 11 days after onset. Recovered.	Yes	No	No bacteria	Distended, gas, gangrene, abscess.
14	Stevenson, 1940	52, M	No	None	X-ray	X-ray, bedrest, sulfathiazole, and antisera. Recovered.	Yes	Yes	No culture	
15	Pierini, 1945	63, F	No	No note	X-ray	Cholecystectomy distal third. Recovered.	Yes	No	B.coli, Staph.albus	Distended, gas, gangrene, abscess.
16	Hutchinson, 1946	56, M	No	Present	Operation	Cholecystectomy 3 days after onset. Recovered.	No	No	Cl.welchii	Distended, gas, gangrene.
17	Culver and Kline, 1948[a]	60, M	Yes	No note	X-ray	Penicillin and sulfadiazine. Recovered.	No	Yes	No culture	
18	Heifetz and Senturia, 1948	57, F	No	Present	X-ray	Cholecystectomy 15 days after onset. Recovered.	Yes	No	Cl.filiforma	Gas, gangrene, abscess.
19	Heifetz and Senturia, 1948	52, M	No	Present	X-ray	Cholecystostomy 22 days after onset. Recovered.	Yes	Yes	Str.fecalis, gram negative rods, spores.	Gas, gangrene, abscess.
20	Friedman et al., 1949	53, F	No	Present	X-ray	Cholecystectomy 12 days after onset. Recovered.	No	No	Staphylococci (no anerobe)	Distended, gas, purulent.
21	Friedman et al., 1949	65, F	Yes	Present	X-ray	Conservative with streptomycin. Recovered.	Yes	No	No culture	
22	Friedman et al., 1949	68, M	No	No note	X-ray	Conservative. Recovered.	Yes	No	No culture	
23	Friedman et al., 1949	66, M	Yes	No note	X-ray	Conservative. Recovered.	Yes	No	No culture	
24	Jemerin, 1949	68, F	Yes	Present	X-ray	Cholecystectomy 16 days after onset. Died.	Yes	No	Cl.oedematiens	Acute, gas, gangrene, abscess.
25	Elsey and Hudson, 1950	75, M	No	No note	X-ray	Cholecystostomy 4 days after onset. Recovered.	No	No	Cl.welchii	Acute, gangrene, abscess.
26	Retterbush, et al., 1951	65, M	No	Present	X-ray	Cholecystectomy 2 days after onset. Recovered.	No	No	B.coli	Acute, distended, crepitation, abscess.

TABLE I (CONTINUED)

CASES OF EMPHYSEMATOUS CHOLECYSTITIS COMPILED FROM THE WORLD LITERATURE

Case	Author and Year	Age,Sex	Diabetes	Calculi	Diagnosis	Treatment and Result	Mass	Jaundice	Culture	Pathology
27	Gowdey and Copeland,1950	57,M	No	Present	X-ray	Cholecystectomy 5 days after onset. Recovered.	No	No	Gram positive and gram negative bacilli (gas-forming)	Acute,distended,gas,gangrene.
28	Mooney and Matzinger,1952	70,M	No	Present	X-ray	Cholecystectomy 24 days after onset. Recovered.	No	No	Not done	Acute,gangrene,abscess.
29	Bell et al.,1953	56,M	No	None	X-ray	Cholecystostomy 5 days after onset. Recovered.	No	Yes	No growth	Distended,gas,gangrene.
30	Bell, et al.,1953	55,M	No	Present	Operation	Cholecystostomy 5 days after onset. Recovered.	Yes	No		Acute,Distended,gangrene,abscess, peritonitis.
31	Jacobs,1956	64,F	Yes	None	X-ray	Cholecystectomy 18 days after onset. Recovered.	No	No	B.coli	Distended,gas.
32	Jacobs,1956	75,F	Yes	Present	X-ray	Fundectomy 8 days after onset. Recovered.	No	No	Staphylococci	Distended,gas,abscess.
33	Quist,1951	63,M	No	Present	X-ray	Cholecystostomy 6 days after onset. Recovered.	No	No	No anaerobic culture	Acute,gangrene,abscess.
34	Rothbart and Steinberg,1952	63,F	No	Present	X-ray	Cholecystostomy 12 days after onset. Recovered.	No	No	B.coli and staphylococci	Gas,gangrene,abscess.
35	Rothbart and Steinberg,1952	51,M	No	Present	X-ray	Cholecystostomy 3 days after onset. Recovered.	Yes	No	Lost	Distended,gas,gangrene,pus.
36	Rocco et al.,1952	45,F	No	Present	X-ray	Conservative 15 days. Readmitted and operated after 6 weeks. Recovered.	No	No	B.coli,Streptococci, and gram positive rods	Acute,distended,abscess.
37	Schwinger et al.,1952	74,F	Yes	No note	X-ray	Conservative:penicillin and streptomycin. Recovered.	No	No		
38	Abel and Rousselot,1952	44,F	No	Present	X-ray	Cholecystectomy 12 days after onset. Recovered.	Yes	No	Negative culture	Acute,distended,abscess.
39	Ryan et al.,1953	70,M	No	Present	X-ray	Cholecystectomy 3 days after onset. Recovered.	No	No	Gram positive bacilli (anerobic gas forming)	Distended,gas,gangrene.
40	Rizk and Anderson,1955	67,M	No	Present	X-ray	Cholecystectomy 12 days after onset. Recovered.	No	No	Staph. aureus	Acute,distended,gangrene.
41	Lacour and O'neil,1956	67,M	Yes	No note	Operation	Cholecystectomy 2 days after onset. Recovered.	No	No	Cl. perfringens	Acute,gas,gangrene.
42	Tedesco,1951	64,F	No	Present	X-ray	Cholecystostomy after 4 weeks. Recovered.	No	No	B.coli and anaerobes	Acute,distended,gas,abscess.
43	Phillips,1954	65,M	No	No note	X-ray	Cholecystostomy after more than 2 weeks. Recovered.	Yes	Yes	Hemolytic staph. aureus. No anaerobes	Acute,tense,abscess.
44	Bosch delMarco,1954	63,M	No	None	X-ray	Cholecystostomy 5 days after onset. Recovered.	No	Yes	Anaerobic gram positive bacilli	Acute,distended,gangrene,abscess.
45	Stromme,1955	27,M	No	No note	X-ray	Conservative:penicillin. Recovered.	No	No		
46	Tooms and Barlow,1955	63,M	Yes	No note	X-ray	Conservative. Recovered.	Yes	Yes		
47	Olander and Bowman,1956	65,M	No	Present	X-ray	Cholecystectomy after 3 weeks. Recovered.	No	No		Gangrene,abscess,free perforation.
48	Olander and Bowman,1956	64,M	No	None	X-ray	Cholecystectomy after more than 7 days.Recovered	Yes	No	Cl.welchii	Acute,distended,gangrene,abscess.
49	Case,1957	57,M	No	Present	X-ray	Cholecystostomy after 15 days. Recovered.	Yes	No	Staph.aureus hemolyticus	Subacute,necrosis,abscess.
50	Edinburgh and Geffen,1957	62,F	No	Present	X-ray	Conservative. Cholecystectomy after 6 weeks. Recovered.	No	Yes	B.coli,B.lactis aerogenes, B.pyocyaneus	Acute,distended,gangrene,abscess.
	*Same case as reported by Brown and Milch.									

rigidity in the right upper quadrant of the abdomen, and a palpable mass is noted in twenty-two or 44 per cent of cases. The white blood cell count ranges from 10,000 to 30,000 per cu. mm. with an increase in polymorphonuclear leukocytes and immature forms. There is the picture of increasing toxicity in the untreated case.

Frequently, a previous history of colic, or of gallbladder disease, varying from a couple of weeks to thirty years in duration, is reported. Although acute cholecystitis appears to favor the females in the ratio of 82 per cent to 18 per cent over the males [*27*], in this process the males predominated over the females at 36:14 or 72 per cent males to 28 per cent females. The age range was thirty-two to seventy-five years, with an average of about fifty-nine years, and more than 80 per cent of patients were past fifty years of age. Diabetes was definitely established in twelve of fifty patients or in 24 per cent. Jaundice was reported in 20 per cent (ten cases). Calculi were found in twenty-nine patients (seven patients had no calculi) or 80 per cent of patients in which there was any report on calculi.

Diagnoses. Obviously the clinical picture, in itself, does not present any features distinctive enough to enable us to diagnose this condition apart from an acute cholecystitis. The x-ray examination of the abdomen, as first described by Hegner [*15*] in 1931, provides the only method of making a specific preoperative diagnosis of acute emphysematous cholecystitis. Therefore, it is obvious that x-ray examination of the abdomen, with special films of the right upper quadrant (posterior-anterior and erect, or lateral recumbent), is of great value in patients presenting with the clinical picture of acute cholecystitis, or with an obscure acute abdominal problem.

The diagnosis was made by x-ray findings in forty-three cases in which preoperative films were taken, and in the other seven cases the diagnosis was made at operation. In three of the patients in whom diagnoses were made at operation preoperative films were diagnostic but were not recognized at the time. The remaining four patients had no preoperative x-ray examination of the abdomen. Therefore, we see that the diagnosis was made or could have been made on the basis of roentgenograms of the abdomen in all cases in which these were taken.

An abdominal film shows gas in the lumen of the gallbladder, in its walls, or in the pericholecystic tissues. Frequently no gas is seen until twenty-four to forty-eight hours after the onset of the acute attack; serial films are therefore of great significance [*30*]. Initially the plain film may show a large, globular gas-filled gallbladder with a fluid level (on the upright view). Later, there appears a darker, concentric annular shadow of lesser gaseous density which represents gas within the wall of the gallbladder, generally in the submucosal layer, thus stripping the mucosa from the overlying wall. Still later, the gas apparently is absorbed within the lumen [*11*], because the concentric, smooth, clearly demarcated ring surrounding the gallbladder is replaced by a streaked and bubble-like appearance representing a mixture of gas and fluid. The vacuolized shadows establish the presence of gas bubbles in the surrounding tissues, sometimes even in the ducts [*11,35*]. If recovery occurs without an operation the amount of gas diminishes and the gallbladder decreases in size. However, small amounts of gas may persist even after the clinical attack has subsided [*30,35*].

Differential Diagnosis. The appearance of the distended gallbladder with gas in the lumen, and also circumferentially within the wall, is diagnostic. However, there are other conditions which may result in gas in the gallbladder, and therefore must be considered in the differential diagnosis: (1) An internal biliary fistula, due either to pathological or operative causes. In these instances, gas is also seen in the biliary radicles, the gallbladder may not be distended, and there is no gas in the wall of the gallbladder, nor any fluid level within the lumen of the gallbladder. The communication to the gastrointestinal tract may be demonstrated by barium meal or by barium enema. (2) Lipomatosis of the gallbladder. The radiolucency of lipomatous tissue may simulate gas in the gallbladder; however, there is no fluid level and no change in the configuration of the translucent shadow with change in position. (3) Incompetence of the sphincter of Oddi may result in gas within the gallbladder, but the ducts should then be outlined with gas as well [*24*].

Operative Findings and Pathology. At operation there is usually a tense, distended gallbladder with acute and chronic changes in the

walls. There is gas under pressure in the lumen and a foul-smelling or purulent exudate. Often there is a pericholecystic abscess, and many dense and filmy adhesions obliterating the anatomy of the area. Frequently the mucosa is gangrenous and completely separated from the muscularis. Biliary calculi are present in the great majority of gallbladders (in more than 80 per cent of patients), but we encountered no description of stones in a pericholecystic abscess. This may be due to incomplete descriptions, or to lack of a gross perforation, as in our case wherein although innumerable small stones were present in the gallbladder, none were found in the abscess.

Pericholecystic abscess is a very frequent occurrence—in twenty-seven of thirty-eight patients operated upon (71 per cent)—and might be considered part of the pathological process if it is allowed to fully develop. On serial x-ray studies it has been shown that gas spreads beyond the confines of the gallbladder before resolution occurs [*2,9,11,17,22,30,44,45*], indicating perforation with abscess formation [*8,38*]. Gas from the lumen apparently enters the pericholecystic tissues via local areas of necrosis in the mucosa and submucosa [*32*]. Experimentally it has been demonstrated [*16*] that with progressive distention of the diseased gallbladder with air, there results a leakage of air into the perimuscular layer, in the region of the occluded cystic duct; nearly the entire submucosal layer is elevated by air before rupture into the pericholecystic tissues occurs. Usually there is a localized, walled-off abscess (as in our patient), but occasionally a diffuse peritonitis can occur [*2,8,30,32*]. Even if perforation has not occurred, the gangrenous changes in the gallbladder wall may be so extensive that it actually fragments during removal [*37*]. In our case the process had actually dissected a plane between the layers of the gallbladder wall so that a gangrenous mucosal cast of the gallbladder was removed. This has been noted by other workers several times in the past. Similarly, often a gangrenous mucosa completely separated from the muscularis has been described [*37*], and this explains the x-ray delineation as a concentric ring of lesser density. The thickened wall may feel crepitant. Only occasionally will gas be demonstrated in the gallbladder wall microscopically [*42*].

Therapy. If we review the previous cases, the treatment and results, we could reach some conclusion as to the ideal methods of treatment.

Twenty-three patients were treated by cholecystectomy from five hours to twenty-four days after the onset of symptoms; there were three deaths, two in the preantibiotic era [*8,15*] and one [*22*] was due to pulmonary emboli in a patient undergoing interval cholecystectomy after fourteen days of conservative treatment. Eight patients were treated in the preantibiotic era with cholecystectomy alone, and two deaths occurred, but of the other fifteen treated with cholecystectomy and antibiotics there was only one death due to pulmonary embolus [*22*].

Fourteen patients were treated with cholecystostomy and one death occurred [*30*], and this was in the preantibiotic era and was due to an overwhelming sepsis with edema and emphysema of the wound, abdominal and chest walls. The other thirteen patients were treated in the antibiotic era with cholecystostomy and antibiotics, surgery being performed three to twenty-eight days after onset and antibiotics administered for an average of seven days preoperatively; all survived.

Thirteen patients were treated conservatively, only one of whom was treated in the preantibiotic era, and all recovered.

A pericholecystic abscess was encountered in twenty-seven of thirty-eight patients operated upon after an interval of two or more days from the onset of symptoms. Of the eleven patients in whom no abscess was found at operation seven were operated upon in three days or less from the onset of symptoms; of the other four patients, two were operated upon five days [*2,14*], one eighteen days [*21*], and one twelve days [*11*] after the onset of symptoms. Apparently the probability of a pericholecystic abscess occurring was greatest about three or more days after the onset of the attack.

Our patient was first diagnosed after a five-day history and at that time there was already air in the wall of the gallbladder. The patient appeared to withstand her acute infection well with the aid of large doses of antibiotics; operation was deferred, and following subsidence of the acute phase (two weeks after the onset) the patient was sent home with instructions to return one month later to undergo an interval operation. The opera-

tion essentially consisted of removing the demarcated mucosal cast of the gallbladder and draining both the pericholecystic abscess and the cystic duct stump. Specific antibiotics were used postoperatively without the aid of specific gas gangrene antitoxins. Although some surgeons [*21*] have used polyvalent gas bacillus antiserums as an adjunct in treatment there is no good evidence that this is necessary.

We may conclude then that the ideal treatment is cholecystectomy if the patient is seen within the first forty-eight to seventy-two hours after the onset of symptoms, supplemented with the use of penicillin and broad-spectrum antibiotics [*31*]. Later gas spreads beyond the confines of the gallbladder indicating that perforation and usually a localized pericholecystic abscess has occurred. Therefore, if the patient is first seen after a lapse of two or three days it would probably be wisest to try the effects of antibiotic therapy, and if the process appears to subside, to wait six to twelve weeks to operate in a symptom-free period. If, on the other hand, the clinical conditions worsen under antibiotic therapy, surgery may still become imperative. Under those circumstances, if cholecystectomy is considered unwise because of the local pathology, then cholecystostomy or hemicholecystectomy with drainage and antibiotic coverage should lead to a satisfactory result.

SUMMARY

Acute emphysematous cholecystitis is a rare pathological condition of the gallbladder characterized by the growth of gas-producing organisms within a gallbladder, the seat of an acute cholecystitis. The presenting clinical picture is usually indistinguishable from that of an acute cholecystitis. The diagnosis is actually made by roentgen studies of the abdomen which demonstrate a distended gallbladder with air in its lumen and within its wall. The most frequent pathological findings are an acutely inflamed and distended gallbladder containing calculi, usually with gangrenous and suppurative changes, and frequently complicated by a pericholecystic abscess.

We may conclude that operation within the first twenty-four to seventy-two hours after onset of symptoms with cholecystectomy, if technically feasible, is the treatment of choice. However, conservative management with the use of large doses of antibiotics has led to satisfactory results in the few patients so treated, and therefore, those that prefer to adopt that method of therapy might be justified in so doing.

Acknowledgment: The authors are indebted to Dr. Arthur Fishberg and Dr. Leon Ginzburg for permission to report this case.

REFERENCES

1. Abel, G. and Rousselot, L. M. Pneumocholecystitis. *New England J. Med.*, 246: 662, 1952.
2. Bell, L. G., Brown, R. B. and Lenhardt, H. F. Acute pneumocholecystitis; review and report of two cases. *Ann. Surg.*, 138: 268, 1953.
3. Bosch del Marco, L. M. Pio neumocolecistitis aguda. *Bol. Soc. cir. Uruguay*, 25(5): 503, 1954.
4. Brown, R. K. and Milch, E. Gallbladder gas gangrene. *Gastroenterology*, 10: 626, 1948.
5. Case, T. C. Acute pneumocholecystitis. *Am. J. Surg.*, 93: 457, 1957.
6. Cottam, G. G. Gangrenous cholecystitis. *Surg., Gynec. & Obst.*, 25: 192, 1917.
7. Culver, G. J. and Kline, J. R. Acute gaseous cholecystitis. *Radiology*, 50: 536, 1948.
8. Del Campo, J. A. and Otero, J. P. Neumotoxis espontinea de las vias biliares. *Arch. urug. med.*, 17: 341, 1940.
9. Elsey, E. C. and Hudson, H. C. Acute gaseous cholecystitis. *Am. J. Roentgenol.*, 63: 228, 1950.
10. Friedman, C. and Poster, H. Emphysematous cholecystitis. *New York J. Med.*, 55(12): 1720, 1955.
11. Friedman, J., Aurelius, J. R. and Rigler, L. G. Emphysematous cholecystitis. *Am. J. Roentgenol.*, 62: 814, 1949.
12. Gordon-Taylor, G. and Whitby, E. H. Bacteriological studies of fifty cases of cholecystectomy with special reference to anerobic infections. *Brit. J. Surg.*, 18: 78, 1930.
13. Gordon-Taylor, G. and Whitby, E. H. Incidence of anerobic infections in the gallbladder. *Brit. J. Surg.*, 19: 619, 1932.
14. Gowdey, J. F. and Copeland, N. N. Acute gaseous cholecystitis. *New England J. Med.*, 242: 647, 1950.
15. Hegner, C. F. Gaseous pericholecystitis with cholecystitis and cholelithiasis. *Arch. Surg.*, 22: 993, 1931.
16. Heifetz, C. J. and Wyloge, E. I. Effect of distention of gallbladder with air and its relationship to acute pneumocholecystitis. *Ann. Surg.*, 142(2): 283, 1955.
17. Heifetz, C. J. and Senturia, H. R. Acute pneumocholecystitis. *Surg., Gynec. & Obst.*, 86: 424, 1948.
18. Hoback, W. W., Cunningham, D. A., McCue, F. A. and Vermilya, G. D. Acute pneumocholecystits; case report. *Am. Surg.*, 20: 1199, 1954.
19. Holle, M. and Marquezy, M. Cholecystite perforante et gangreneuse. *Bull. et mém. Soc. méd. hôp., Paris*, 46: 49, 1922.
20. Hutchinson, W. R. S. Gas gangrene of gallbladder. *Brit. M. J.*, 1: 915, 1946.

21. JACOBS, J. K. Acute emphysematous cholecystitis. *Am. J. Surg.*, 91: 799, 1956.
22. JEMERIN, E. E. Cholecystitis, emphysematosa. *Surgery*, 25: 237, 1949.
23. KENT, P., II. Acute Pneumocholecystitis. *Gastroenterologia*, 76: 6, 350, 1950.
24. KERLEY, P. A. Gallbladder visualized. *Brit. J. Radiol.*, 13: 289, 1940.
25. KIRCHMAYR, L. Gas gangrene of gallbladder in elderly man. *Zentralbl. Chir.*, 52: 1522, 1925.
26. KU, Y. C. Acute pneumocholecystitis with report of a case. *Chinese M. J.*, 68: 9–10, 371, 1950.
27. LACOUR, E. G. and O'NEIL, L. J. Acute pneumocholecystitis; a review of the literature and report of one case. *Surgery*, 39: 813, 1956.
28. LOBINGER, A. S. Gangrene of the gallbladder. *Ann. Surg.*, 48: 72, 1908.
29. MOONEY, B. R. and MATZINGER, K. E. Acute gaseous cholecystitis. *Canad. M. A. J.*, 66: 66, 1952.
30. MCCORKLE, H. and FONG, E. E. Clinical significance of gas in the gallbladder. *Surgery*, 11: 651, 1942.
31. NEWTON, A., et al. Sensitivities of ten species of clostridia to penicillin, aureomycin, terramycin, and chloramphenicol; study of battle wounds in Korea. *Surgery*, 37: 392, 1955.
32. OLANDER, G. A. and BOWMAN, J. A. Acute pneumocholecystitis; a review and report of two cases. *Arch. Surg.*, 72: 709, 1956.
33. PHILLIPS, S. H. Acute pneumocholecystitis; a case report. *J. Indiana M. A.*, 47: 249, 1954.
34. PIERINI, A. Gangrena gaseosa de la vesicula giliar. *Dia méd.*, 17: 988, 1945.
35. PIKOVSKII, D. L. Gazovyi Kholetsistit. *Khirurgiia, Moskva*, 3: 80, 1954.
36. QUIST, C. F. Cholecystitis emphysematosa. *Acta. radiol.*, 35: 200, 1951.
37. RETTERBUSH, W. C., FISHMAN, M. B. and BAIRD, W. A. Emphysematous cholecystitis; report of case with successful treatment. *Ann. Surg.*, 134: 268, 1951.
38. RIZK, W. S. and ANDERSON, A. E., JR. Emphysematous cholecystitis. *J. Florida M. A.*, 42: 384, 1955.
39. ROCCO, A. F., HUNT, R. R. and SAIRAN, J. Acute pneumocholecystitis. *Radiology*, 58: 228, 1952.
40. ROTHBART, F. and STEINBERG, M. J. The preoperative diagnosis of gaseous inflammations in the acute abdomen. *J. Internat. Coll. Surgeons*, 18: 349, 1952.
41. RYAN, E. A., HARRIGAN, E. and PENNY, S. F. Acute gaseous cholecystitis. *Canad. M. A. J.*, 69: 606, 1953.
42. SCHMIDT, E. A. Emphysematous cholecystitis and pericholecystitis. *Radiology*, 31: 423–427, 1938.
43. SCHOTTENFELD, L. E. Anerobicinfections of the biliary tract. Review and eight new cases. *Surgery*, 27: 701, 1950.
44. SCHWINGER, A., HEMLEY, S. D. and HARRINGTON, L. A. Pneumocholecystitis. *Gastroenterology*, 22: 272, 1952.
45. SIMON, J. Le pyopneumocholecyste et son diagnostic radiologique. *Presse méd.*, 40: 1932, 1938.
46. STEVENSON, C. A. Emphysematous cholecystitis. *Am. J. Roentgenol.*, 51: 53, 1944.
47. STOLZ, A. Ueber Gasbildung in den Gallenwegen. *Virchows Arch. path. Anat.*, 165: 90, 1901.
48. STROMME, A. Emphysematous cholecystitis; report of a case. *Acta radiol.*, 44(1): 39, 1955.
49. TANNER, E. K. Acute gas producing cholecystitis. *Surg., Gynec. & Obst.*, 32: 468, 1921.
50. TEDESCO, B. Pyo-pneumo-cholecyste; contribution à l'étude de la cholecystite gangreno-gazeuse. *Arch. mal. app. digest.*, 40: 7–8, 906, 1951.
51. TOOMS, D. and BARLOW, D. Acute emphysematous cholecystitis. *Proc. Roy. Soc. Med.*, 48(9): 757, 1955.
52. WYBAUW, L. Image vésiculaire inhabituelle. *J. belge Gastro-entérologie*, 4: 123, 1936.

Vaginal Plastic Surgery Based on a New Concept of the Anatomy of the Female Genital Tract

VI. SURGICAL TREATMENT OF THE LACERATED PERINEUM OF LONG-STANDING*

HERMAN SHANN, M.D., *Brooklyn, New York*

LACERATION of the perineum, whether of the first or second degree, is essentially an asymptomatic condition, so far as the patient is concerned. In fact, she may not even be aware of it until she is told by a physician that she was "torn." That, presumably, is what TeLinde had reference to in his discussion of vaginal plastic operations as an "asymptomatic relaxation" [3]. Injuries above the perineum are, of course, not asymptomatic.

The old concept that lacerations of the perineum are a direct cause of displacements and prolapse of the uterus can no longer be tenable, if we take cognizance of the fact that when laceration of the perineum to an extreme degree occurs, that is, of the third degree or an anorectal laceration, prolapse of the uterus is not known to occur even in neglected cases of long-standing.

The third degree laceration, better designated as the anorectal injury, was fully considered in a previous article of this series [1]. It was dealt with as a distinct and separate entity, that is, as an injury affecting a part of the intestinal tube, and its treatment received special attention.

However, so far as the less severe lacerations of the perineum are concerned, their repair, if undertaken at all, must have as an objective the restoration of a normal anatomic organ for cosmetic effect or to favor conjugal felicity. The usual practice of perineoplasty, as described in textbooks, consists of the excision of so much "redundant" vaginal mucosa and tightening up the orifice by "high perineorraphy." This results in a distorted, inelastic tube and in most cases is useless as a functional organ.

To reconstruct a normal anatomic vagina, the natural constituent elements of the perineum must each receive special attention. The application of certain surgical principles will then yield better operative results.

SURGICAL PRINCIPLES OF PERINEOPLASTY

The general surgeon, in undertaking the treatment of the fracture of a bone, has to observe a certain well established principle, namely, that the first element in the reduction of the fracture is to reproduce the fracture as it originally occurred at the moment of injury, and then apply whatever treatment may be indicated in the particular case.

The same principle should be adopted in the treatment of a "fracture" of the perineum. (This term is temporarily substituted for rupture of the perineum, to emphasize the analogy of the two conditions regarding the principle involved in treatment.) The original wound as it occurred at the time of delivery should first be reproduced, followed by surgical repair.

When the perineum is ruptured during delivery, the gaping wound forms an acute angle: the apex below and the torn tissues form the sides of the angle.†

† The laceration is not always a sharp, straight line, but the basic principles in treatment are always the same.

* Parts I, II, III, IV and V appeared in the May, July and October 1957 issues, and the March and May 1958 issues, respectively, of *The American Journal of Surgery*.

If the perineal laceration is not repaired immediately and the wound is allowed to heal by scar, certain changes will inevitably develop which are characteristic in all perineal lacerations of long-standing.

As a result of the laceration, a certain muscular element is ruptured and its torn ends retract in opposite directions. The angle of the wound widens and will gradually become flattened out, to become eventually a straight transverse line. When the wound finally heals by scar, a widened orifice of the vagina and a shortening of the posterior vaginal wall results. In some cases there is so much shortening of the posterior vaginal wall that the cervix appears to be close to the orifice. The condition is erroneously regarded as a first degree prolapse of the uterus, when there is no prolapse at all.

On the other hand, if the perineum is repaired immediately by the obstetrician, but, due to haste or exigency of the case, the sutures take in only vaginal wall and do not "catch" the torn musculature, the results will not be any better. Here, too, retraction of the torn ends of the muscle lead to a widening of the orifice, a shortening of the posterior vaginal wall and exposure of the cervix, which is usually observed in many parous women.

In the repair of perineal lacerations, two important anatomic constituents have been entirely ignored, namely, the sphincter vaginae muscle and the fascia intimately associated with it. The utilization of these two important structures will be particularly stressed in the operative technic of perineoplasty, to be described.

OPERATIVE TECHNIC OF PERINEOPLASTY

The mucocutaneous junction is incised, as is described in textbooks, but the scalpel is used instead of scissors as the latter causes distortion and needless sacrifice of tissue. In some cases the incision must be quite deep in order to reach and expose the original inner structures of the perineum.

The vaginal wall is separated for about 1 cm. from the deeper structures in front of it. The skin, likewise, is separated from the structures behind it. There will now be exposed from within, outward, three layers of tissue, namely, the vaginal wall, the middle layer of the deep perineal structures and, finally, the skin.

To reproduce the original angularity of the wound, the middle layer in the median line is grasped with a tenaculum and, as traction is made downward, the straight line of the wound is now converted into an angular wound which simulates the laceration as it originally occurred at the time of delivery. This brings the sides of the angle close to the median line and in some cases may even present in sharp outline the constituent elements of the middle layer.

This successful result may not be attained in every case, as when much atrophy or sloughing of the tissues has occurred as a result of infection, and also when much scarring has taken place. In all such cases considerable dissection will be required to expose the individual elements.

In repairing the individual layers, one starts with the vaginal wall. A few interrupted submucous sutures bring the sides together in the median line. A clamp on the uppermost suture, held on tension by an assistant, brings out in sharp outline the next layer of tissues which consists of the sphincter vaginae muscle and the fascia. If the muscle has not yet been visualized, the fascia is dissected up for some distance until the muscle is exposed. The fascia is invariably exposed as soon as the sides of the vaginal wall are brought together.

REPAIR OF THE SPHINCTER VAGINAE MUSCLE

This type of tissue must receive special attention. If catgut suture is used, the chances are that in absorbing moisture it becomes soft and loose, and the muscle ends will retract again. This will defeat the purpose of the operation. It is advisable, therefore, to use very fine silk as suture material, and mattress stitch the torn ends. This will prevent retractions and will favor good healing.

The next step is closure of the fascia with interrupted chromic sutures, which take a few bites in the underlying muscle, to splint it and hold it in place. The skin wound is then closed with a few interrupted silk sutures.

The vaginal mucosa may be closed with a continuous chromic suture; if it has been perfectly approximated by the submucous sutures, it needs little further attention. Mucous membranes, if closely approximated and if perfect hemostasis has been practiced, heal very rapidly, almost overnight, without any sutures. This is good, for infection may be introduced through each stitch hole.

How high should the perineum be built up?

The guide is the upper border of the sphincter vaginae muscle.

The levator ani muscle is not dragged into the wound. It does not belong there and will not stay there. In bringing the torn fascia together in the median line, the levator muscles will be restored to their natural anatomic position.

A piece of iodoform gauze is left in the vagina to absorb moisture, and is removed in twenty-four or forty-eight hours.

COMMENTS

In the previous article dealing with anorectal injury [1], closure of the perineal part of the wound was not discussed. This was intentionally deferred until the present article on perineal repair was completed. It must be quite apparent that the same principles are applicable in anorectal-perineal repair. However, since the wound is more extensive, more care will be necessary in isolating the several layers of tissue and bringing them together in their normal anatomic relationship.

It would be presumptuous of me to suggest even for a moment that all problems of vaginal plastic surgery have been solved in this series of articles.

Let it be understood that the chief object has been to present a concept of the female genital tract which is at considerable variance with the usual textbook descriptions and is based upon personal experience and observations over many years of practice in this field. Furthermore, as a result of extensive study, certain pathologic conditions resulting from parturition have received a different interpretation.

That the results of the usual plastic operations, as described in textbooks, have been disappointing has been known to every surgeon of experience. Recently, however, this has been evidenced by frank expressions in the gynecologic literature. We have, for instance, the idea conveyed by Meigs [2] who states that: "Another glance at Table 1 will illustrate the change in gynecologic surgery in our hospital; vaginal plastic operations being replaced by vaginal hysterectomy."

Then, again, TeLinde, in his discussion of a paper by Wilson [3] before a national society on vaginal plastic surgery, made the following startling remarks: "In my opinion there are thousands of unnecessary operations done for asymptomatic vaginal relaxations. . . . The patient should be told what she may expect from the repair, and she may make up her mind whether she wants it."

The evaluation of these remarks poses this perplexing question: Are we justified in continuing the conventional type of operative procedures, which are still practiced on hundreds of cases daily, when leading surgeons intimate a lack of faith in their efficacy? Should we not look for other remedial and preventive measures which would offer better safeguards for the welfare of the human female?

It must be realized that the only positive preventive of all birth injuries is, of course, cesarean section, and even that can be effective only when done early, that is, before the stress and strain of labor have caused damage to the neighboring pelvic tissues. Admittedly, the time is not yet ripe for the universal adoption of so radical a remedy. But is it not about time to give this matter serious thought?

When a young woman consults an obstetrician for the first time about her pregnancy, she usually knows nothing about the process of parturition. She has no idea as to what is going to happen to her except, perhaps, that she is going to have pain, and she hopes that the doctor will not let her suffer too much. However, so far as the grave injuries she is liable to sustain are concerned, that is, injuries to the upper areas of the genital tract, she has no conception of them and the obstetrician, with his best intentions, cannot do anything to prevent them.

In contrast, there is the mature and otherwise well informed woman who states very definitely that she will have just one baby and no more. Should she not be informed as to what the "natural" delivery may do to her, if she is to go through a difficult labor terminating perhaps in a violent forceps delivery which may wreck her health and destroy her future usefulness, even resulting in permanent invalidism? This may appear exaggerated, but I believe every obstetrician can recall such cases.

These two distinct types of patients have one thing in common, ignorance as to what lies ahead for them if conditions during parturition are unfavorable. Should they not be informed? Should the prerogative of elective cesarean section be limited only to Hollywood personalities or to daughters of nationally prominent citizens?

We have been habituated to the idea that cesarean section is a dangerous operation,

carrying with it a high mortality and morbidity. That, of course, was only too true in former years, but the bad results in that era were not due to the operation, *per se*, but to the wrong timing of it. In those days cesarean section was done only as a last resort when it was realized, rather late, that the baby could not possibly be born by the vaginal route. By that time the patient was exhausted physically, mentally and psychically, starved and dehydrated after many hours or even days of suffering, and in many cases already infected. She was then subjected to an abdominal operation under the most unfavorable conditions and, of course, the results were disastrous.

Things are so different today. The indications for cesarean section are constantly being more and more liberalized. The obstetrician is well trained in his profession and is quite competent to determine in advance when section is indicated. He does not allow the patient's general condition to deteriorate before resorting to it. The result has been that thousands of sections are done nowadays over the land and, in the hands of experienced surgeons, without any untoward incidents.

From a surgical point of view, the cesarean section is actually one of the simplest and safest operative procedures in abdominal surgery for, after all, it entails but two simple straight incisions, one into the abdominal wall and the other into the uterus. There is no exploration to be done. No organ other than the uterus is involved. The intestines are tucked away behind the full term uterus and are hardly seen or handled even after the baby is delivered, which eliminates postoperative abdominal distention. The patient is almost completely well twenty-four hours after the operation and is discharged at an early date, with a baby—a happy, contented and grateful woman.

Postoperative complications do not occur in "clean" cases, but every case should be one.

Other elements are deserving of serious consideration. For instance, are normal or natural deliveries entirely harmless? Is the aftermath of these "natural" deliveries—laceration, displacement, prolapse and the mutilated "hypertrophied" cervix, considered a potential cause of malignancy—to be ignored entirely?

Should we not stop to think of the thousands of women who crowd the gynecological clinics and are subjected to all kinds of operative procedures for the correction of various birth injuries?

Finally, we have the cases of chronic invalidism following prolonged difficult labor, especially those patients who were subjected to brutal forceps deliveries and to whom even reparative surgery has not given relief.

Then, again, can the fetal mortality be ignored, especially those of iatric etiology? Can asphyxia neonatorum, caput succedaneum and the various paralyses be regarded lightly? True, most of these conditions clear up in a few days, but in all of them there occurs considerable disturbance of the cerebral circulation and perhaps also direct injury to some cerebral tissue. Who can tell how much effect this will have on the mental and psychic state of the future man?

This next question has no direct bearing on our problem, but it is deserving of consideration. What about the obstetrician himself? In no other department of medicine is a practitioner called upon to sacrifice so much of himself as is the busy and "successful" obstetrician. Night after night (and the demand upon him is no less during the rest of the day) he watches the suffering patient during the "progress" of labor. Of course, he gets hardened to that kind of life but it ultimately leaves its mark. One recalls the sad fate of many a colleague, relatively young men, victims of this grueling branch of medicine.

This is not a plea for cesarean section, nor does it follow that the obstetrician is to try to "sell" cesarean section to his patients. It is a plea for a change of attitude on the part of obstetricians and the medical profession in general with reference to this very serious problem. The public should be better informed as to what "natural" deliveries do to women; the individual patient is entitled to know what she will go through in giving birth to a baby and especially what lies ahead for her if conditions are not favorable. To repeat the remark of TeLinde, with reference to vaginal plastic surgery, "The patient should be told what she may expect . . . and she may make up her mind whether she wants it."

ADDENDUM

Since completing this article and extolling, perhaps with an exaggerated enthusiasm, the virtues of cesarean section and its simplicity

and safety, I experienced a severe jolt when I read the abstract of an article by Epstein [4]. He reports that within a period of three years rupture of the uterus scar had occurred in sixteen cases, and he states that the fact that "rupture of the scar was found once in twenty cases is a cause of much concern." And well it should be.

The author states further that "faulty technique in the closure of the uterus has been suggested as a cause of rupture of the uterus. The sutures might be too tight or too loose." This may be so, but how is the surgeon to gauge that? We are not yet equipped with an electronic robot to guide us in the amount of tension to be used in each individual suture. In my opinion that is not the reason.

As surgeons, we ought to think in terms of anatomy and physiology when attempting to interpret the cause of failure in any operative procedure. With the use of cesarean section there certainly are anatomical and physiological reasons to account for this unfortunate sequela.

1. Anatomical reason: It will be recalled that the musculature of the uterine wall is arranged in three layers: a longitudinal, a circular and an oblique layer. The fibers of the circular layer run in a transverse direction to the line of incision. What happens when these fibers are cut? The cut ends retract promptly in opposite directions. When the through and through or mass suture is used to close the uterine wound, the suture does not "catch" the circular fibers, and they are not included in the suture because they are not there. The result is a thin, weak suture line. Incidentally, I recall that when I was an interne at the old New York Lying-In Hospital many, many years ago I observed the late Dr. Asa Davis use interrupted sutures, in several rows, to close the uterine incision.

2. The suture material: The catgut suture (even if chromicized), in the presence of tissue juices, absorbs moisture and becomes soft, soggy and loose. This permits slight separation of the wound, leading to a weakened scar. Of course, it does not happen in every case, but probably in more than we are aware of.*

* In one case, at autopsy, it was observed that the catgut had been completely digested and only a few knots were floating in the uterine cavity. There was no union of the wound.

The non-absorbable suture is not affected as much by tissue juices. Therefore, silk would be a better suture material. Used as interrupted mattress sutures, in several rows, it should result in a solid, dependable scar.

3. Should the uterine mucosa be included in the suture? Mucosal wounds heal rapidly without being sutured, if properly approximated submucously. On the other hand, when sutured, every stitch hole permits seepage of secretions along the suture tract. In the case of cesarean section, if a slight infection occurs, the patient may be able to overcome the systemic invasion but, locally, microscopic abscesses may form which will cause destruction of a certain amount of muscle tissue and result in a weakened scar.

4. The use of through and through catgut sutures leaves the knots and knot ends sticking out on the surface of the wound. This favors adhesions of the uterus to the anterior abdominal wall. Later on, as the uterus contracts, the serous coat of the uterus, which is a rather firm supporting structure and gives strength to the wound, may be pulled off some part of the muscle and leave a thin, weak scar.

If silk is used as a continuous inverting suture for the serous coat, a fine, smooth suture line will result with less chance of adhesions.

In summary, then, it may be stated that: (1) The through and through, or mass, suture is not likely to produce a strong scar for it does not "catch" an important layer of the uterine musculature. (2) The catgut suture, weakened by tissue juices especially in so vascular an organ as the uterus, may cause a certain amount of separation of the wound and result in a weak scar. (3) Silk would be a better suture material. It should be used as interrupted mattress sutures in several rows. The serous coat should be closed with a continuous inverting silk suture in every case.

REFERENCES

1. Shann, H. Vaginal plastic surgery based on a new concept of the anatomy of the female genital tract. IV. The anorectal birth injuries. *Am. J. Surg.*, 95: 425–428, 1958.
2. Meigs, J. V. Review of gynecology in the last 50 years. *Surg., Gynec. & Obst.*, 100: 517–525, 1955.
3. TeLinde, R. W. Discussion of paper by J. V. Wilson on vaginal plastic operations. *Am. J. Obst. & Gynec.*, 70: 1223, 1955.
4. Epstein, W. A. Disruption of the postcaesarian scar. *J. Mt. Sinai Hosp.*, 24: 97, 1957.

Key Graft Fixation in Mid-tarsal Fracture Dislocation

ELIHU FRIEDMANN, M.D., *Scranton, Pennsylvania*

GROSS disruption of foot architecture often follows a fall from considerable height. The impaction and fragmentation that results presents a troublesome orthopedic problem. A technic is presented for dealing with the derangement of the mid-tarsal area with splitting of the navicular bone.

MECHANISM OF INJURY

Falls from a considerable height usually cause tarsal disruption when the patient lands on his feet. The talus crushes the os calcis beneath it, producing impaction and comminution in these bones. However, if a portion of this force is directed anteriorly, the convex anterior articular surface of the talus is driven into the concavity of the articulating navicular, thereby splitting this bone and causing additional disruption in the mid-tarsal relationships. (Figs. 1 and 2.)

SURGICAL TECHNIC

In the case presented herein, conventional triple arthrodesis was performed to achieve bony union in satisfactory position between the calcaneus and the talus as well as the calcaneus and the cuboid. However, in order to bridge the comminuted fragments in the mid-tarsal area a graft was taken from the subcutaneous portion of the tibia measuring 1½ by ¾ by ¼ inch. A slot was prepared extending from the anterior portion of the talus through the fragmented navicular into the first cuneiform bone. The graft was carefully impacted to this prepared slot.

CASE REPORT

C. G. (No. 60317), a forty-one year old man was admitted to Moses Taylor Hospital on April 10, 1956. He had fallen 32 feet from a roof while repairing a television antenna and suffered a

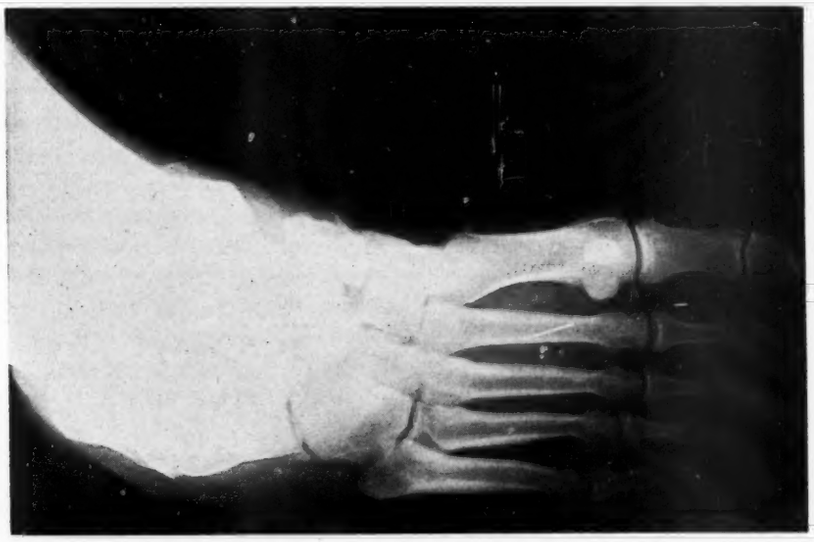


FIG. 1. Oblique view of the right foot, April 10, 1956, showing the comminuted fracture of the navicular bone with anterior displacement of the talus. The broken needle was an incidental finding.

FIG. 2. Lateral view of the right foot, April 11, 1956, after cast application, demonstrating impaction of the talus inferiorly into the os calcis and anteriorly splitting the navicular.

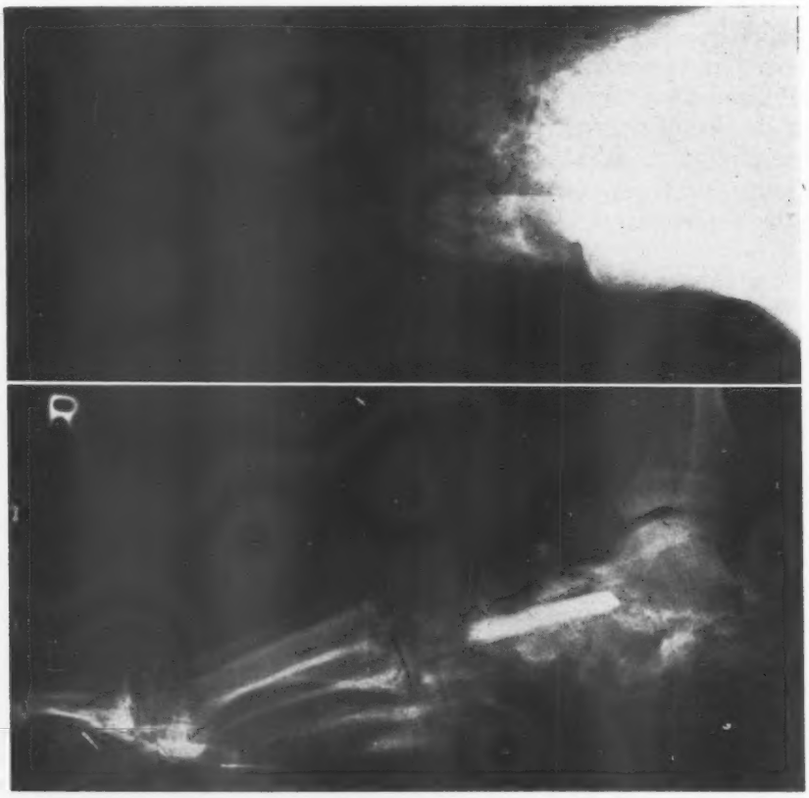


FIG. 3. Anteroposterior (top) and lateral views (bottom) of the foot, October 9, 1956, showing satisfactory healing of both the mid-tarsal and triple arthrodeses.

compound fracture dislocation of the left elbow, a fracture dislocation of the left ankle and a fracture of the right os calcis, talus and navicular. The patient was treated initially for shock and then brought to the operating room where débridement, reduction and primary closure were performed on the elbow. An attempt was made to reduce the leg and foot fractures. All three extremities involved were immobilized in plaster of paris casts. On April 14, 1956, open reduction was performed on the left ankle with screw fixation of the medial malleolus. A satisfactory reduction was then readily obtained and a plaster of paris cast applied. On April 27, 1956, the casts on both legs were changed. On May 1, 1956, the left arm cast was removed, the wound was clean, and mobilization of the elbow was started. The patient was temporarily discharged from the hospital with both lower extremities in casts.

On May 11, 1956, he was rehospitalized at the Hahnemann Hospital where triple arthrodesis was performed along with the placing of a bone graft from the tibia in the mid-tarsal area, as previously described. After four months of immobilization the cast was removed and the foot was found to be clinically solid. X-ray confirmed healing of the triple and mid-tarsal arthrodeses. (Fig. 3.) The patient made excellent progress in the use of both of his lower extremities and on October 29, 1956, was discharged ready to do any type of work. At the time of discharge the patient had full function of the left ankle and left elbow.

SUMMARY

A new technic for mid-tarsal arthrodesis by means of a tibial key graft is presented as a method of dealing with fracture dislocations in this area. In the case cited this procedure was combined with conventional triple arthrodesis.

"Arrived in Paris, Paré became a student and 'companion surgeon' at the Hotel Dieu, the big, dirty hospital close to the Cathedral of Notre Dame on the left bank of the Seine. The Hôtel Dieu, which was founded in 660 A.D., was washed on one side by the evil-smelling waters of the river, which made the building damp the whole year through. The wards were heated by iron stoves, though as one historian remarks, these were scarcely necessary because of the body heat—*la chaleur morbifique*—of the sick. The humid stench crept like a fog out of the curtained beds. Cries, groans, prayers and delirious shrieks filled the halls. As companion surgeon, young Paré must have spent most of his time in the ward to which the wounded and victims of accidents were brought. Some of the survivors of the operating table and women who had been badly injured in childbirth were put there also. Maternity work was in the hands of midwives, who had a ward of their own. It was a sort of cellar, its windows opening just above the stinking waters of the river, which flooded the place several times each winter. Paré would have been summoned down to this stink-hole only when some poor girl was dying in difficult labor, but only then and as a last resort when the midwives could do no more." (From YOUNG, A. Scalpel: Men Who Made Surgery, pp. 33, 34. New York, 1956. Random House.)

Current Therapy of Elephantiasis of the Lower Extremity

THOMAS J. BAKER, JR., M.D., *Columbia, Missouri*

From the Department of Surgery, Section of Plastic Surgery, University of Missouri School of Medicine, Columbia, Missouri.

IN order to understand present day concepts regarding therapy of elephantiasis, a brief historical background will be reviewed.

One of the earliest references to surgical treatment of elephantiasis was by Cornochan [1] in 1851. He treated the condition by ligating the main artery to the limb involved. This was not successful and there was little mention of the subject again until 1908 when Handley [2] theorized that the etiology was an absence of communication between the existing superficial and deep lymphatics. He attempted to connect these by threading pieces of linen thread from the superficial spaces to the deep ones. Four years later he retracted his statements and said the technic was unsatisfactory and should be discarded.

In 1912 the classic article by the Greek physician, Kondoléon [3], was published and this became the first procedure which offered results that were at all encouraging. His technic was to make an incision the length of the leg over its lateral aspect and remove a strip of aponeurosis overlying the muscle. The width of the strip was approximately 3 inches. The skin incision was then closed. Six weeks later he would repeat the procedure over the medial aspect of the leg. This particular technic was expanded and seventy-five cases were treated by Sistrunk at the Mayo Clinic [4]. The method was thereafter referred to as the Kondoléon-Sistrunk procedure in this country. Sistrunk modified the original technic somewhat not only by removing the aponeurosis overlying the muscle but also by excising a similar width of skin and subcutaneous tissue. This helped remove excess bulk.

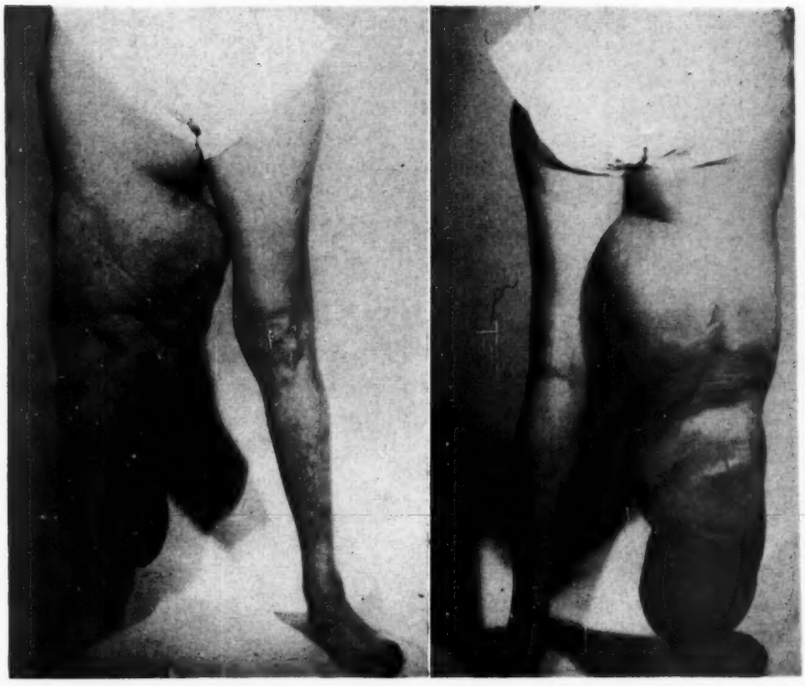
In 1940 Macey [5] reported a procedure which he believed to be an improvement in the management of elephantiasis. He turned back a trap door flap of the involved tissue on the leg, carrying the flap down to and including the fascia of the underlying muscles of the leg. He then covered the raw area on the leg with split-thickness skin grafts and replaced the flap as a dressing. Ten days later he removed the flap and the graft remained as the new epidermal covering for the leg.

In 1947 Poth and co-workers [6] advocated excising the involved tissue and replacing the skin which had been denuded of its subcutaneous tissue, if it was suitable. If not, they utilized split-thickness skin grafts. They made a point of leaving a strip of skin overlying the tibia.

In 1949 Blocker [7] advocated excising all the diseased tissue of the involved extremity including the pretibial strip and resurfacing the denuded area with skin grafts, either from the excised skin if it was suitable or from the opposite leg if indicated.

The histopathological elements necessary for a diagnosis of lymphedema of the lower extremity were outlined in 1913 by Matas [8], who incidentally performed the first Kondoléon procedure in this country.

The necessary criteria as he pointed out are: (1) mechanical obstruction or blockade of the lymphatics or veins, usually from thrombophlebitis, lymphangitis or adenitis; (2) hyperplasia of the collagenous connective tissue of the hypodermis, (3) gradual disappearance of elastic fibers of the skin; (4) a coagulable hard lymphedema; and (5) chronic reticular lymphangitis. He believed the disease was caused by secondary and repeated invasion of pathological microorganisms usually of a streptococcal variety. This theory as to etiology is the one probably most accepted today.



1 2

FIG. 1. Preoperative condition of chronic lymphedema of twenty years' duration.

FIG. 2. Posterior view. Note involvement of entire extremity including the hip.

3 4

FIG. 3. Leg suspended on overhead frame. Area to be excised is marked out.

FIG. 4. Excision of involved tissue is complete. Raw areas will be covered with split-thickness skin grafts after hemostasis is obtained.

FIG. 5. Specimen excised; 6 inch rule gives size relationship. Skin grafts were removed from the nonpigmented area of this specimen.

The following outline is offered as a satisfactory method of managing these cases:

1. The patient must be hospitalized.
2. A complete physical examination is done.
3. A period of at least two weeks of absolute bed rest with the leg elevated is indicated in order to reduce the edema as much as possible.
4. The extremity should be cleansed daily with surgical soap and rid of any infection that might interfere with the "take" of skin grafts.
5. Under general anesthesia the involved areas should be excised down to and including the fascia overlying the muscles.
6. Skin covering the sole of the foot and Achilles tendon should be preserved.
7. The denuded area should be resurfaced with split-thickness skin grafts taken from the opposite leg, abdomen or from the skin which was excised, if it is suitable. Absolute hemostasis is essential.
8. The leg should be carefully dressed and placed in a posterior splint for immobilization.
9. The extremity should be kept elevated for several days to prevent edema and to aid in the take of the grafts.
10. The first dressing is done on the sixth or seventh day.
11. Between the fourteenth and twenty-first day the patient is taken to the whirlpool tank to aid in removing any crusts that remain on the leg and to accelerate the return of function.
12. No walking or standing is permitted until thirty days after the grafts have healed and all raw areas are gone.

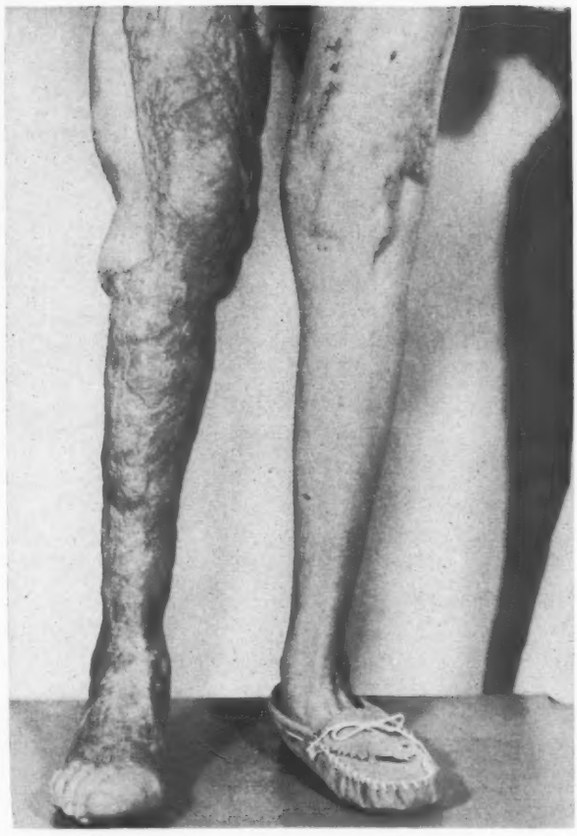


FIG. 6. Result six months postoperatively. Functional result is excellent.

13. Ambulation should be started slowly, with frequent rest periods. Elastic support must be worn constantly.

If these suggestions are carefully carried out, good function as well as satisfactory cosmetic appearance can be attained. (Figs. 1 through 6.)

The patient must understand, however, that he must adjust to a "new way of life" after such an operation and this includes the wearing of elastic support for many months, maybe years; frequent periods of rest with the leg elevated; not remaining in a standing position for prolonged periods of time; and constant care to prevent injury to the limb.

SUMMARY

A brief historical review of surgical treatment of elephantiasis is presented. Present concepts of management are offered, with stress placed on the patient's willingness and ability to adapt to a "new way of life."

A case is photographically demonstrated. showing a satisfactory result.

REFERENCES

1. Cornochan. Quoted by Matas [8].
2. Handley, W. S. Lymphangioplasty. *Lancet*, 1: 783–785, 1908.
3. Kondoléon, E. Die Lymphableitung, als Heilmittel bei chronischen Oedomen nach Quetschung. *München. med. Wchnschr.*, 59: 525–526, 1912.
4. Sistrunk, W. E. Further experiences with the Kondoléon operation for elephantiasis. *J. A. M. A.*, 71: 800–806, 1918.
5. Macey, H. B. A new surgical procedure for lymphedema of the extremities: report of a case. *Proc. Staff Meet., Mayo Clin.*, 15: 49, 1940.
6. Poth, E. J., Barnes, J. R. and Ross, G. T. A new operative treatment for elephantiasis. *Surg., Obst. & Gynec.*, 84: 642–644, 1947.
7. Blocker, T. G. The surgical treatment of elephantiasis of the lower extremities. *Plast. & Reconstruct. Surg.*, 4: 407, 1949.
8. Matas, R. The surgical treatment of elephantiasis and elephantoid states dependent upon chronic obstruction of the lymphatic and venous channels. *Am. J. Trop. Dis. & Prevent. Med.*, 1: 60–85, 1913.

"We tearme sleepe a death, and yet it is waking that kils us, and destroyes those spirits that are the house of life. Tis indeed a part of life that best expresseth death, for every man truely lives so long as hee acts his nature, or someway makes good the faculties of himselfe: *Themistocles* therefore that slew his Souldier in his sleepe was a mercifull executioner, 'tis a kinde of punishment the mildnesse of no lawes hath invented; I wonder the fancy of *Lucan* and *Seneca* did not discover it. It is that death by which we may be literally said to die daily, a death which *Adam* died before his mortality; a death whereby we live a middle and moderating point betweene life and death; in fine, so like death, I dare not trust it without my prayers, and an halfe adiew unto the world, and take my farewell in a Colloquy with God." (From: Browne's Religio Medici and Digby's Observations, pp. 173–174. Oxford, 1909. Clarendon Press.)

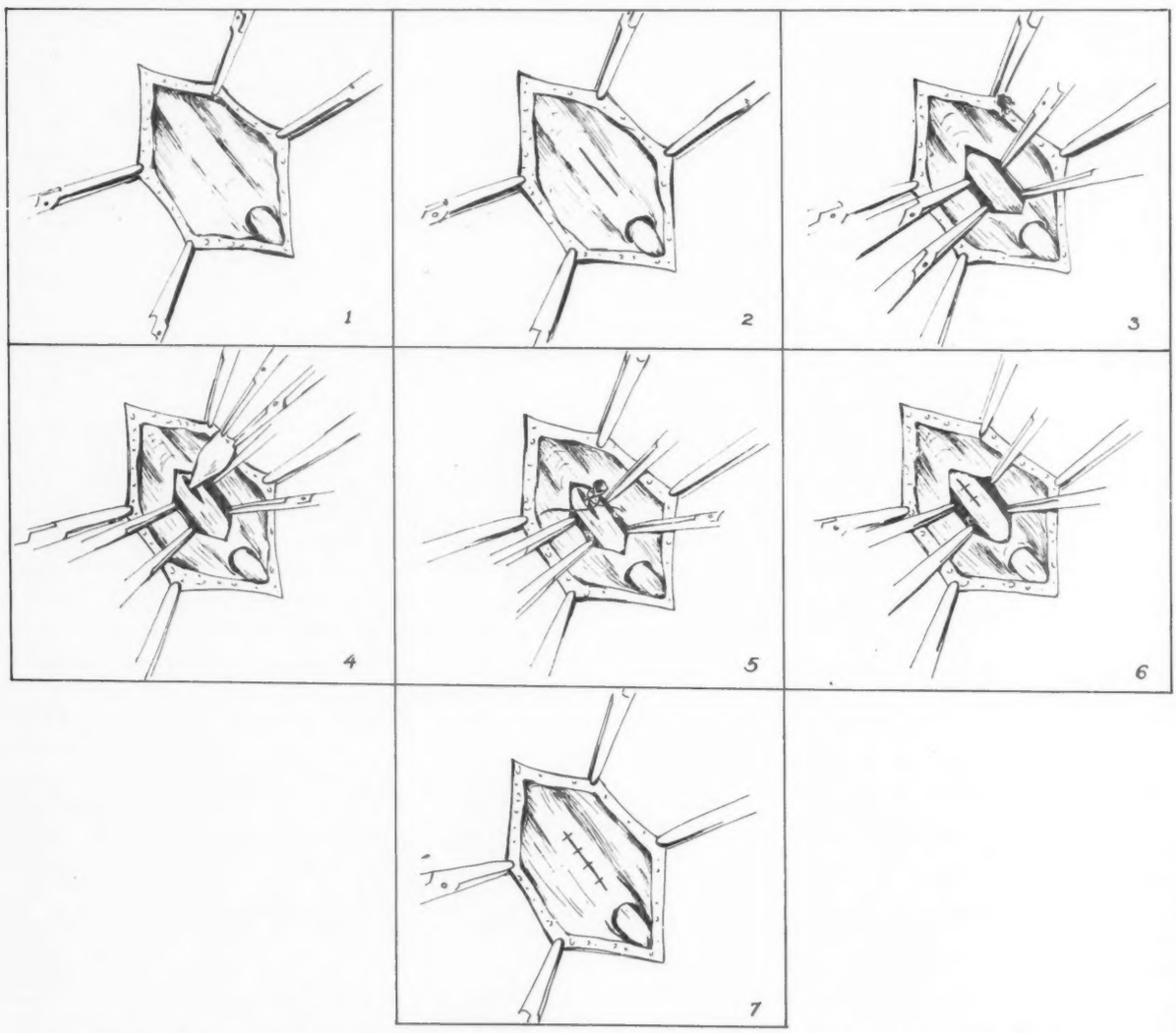
"It would be very convenient for the surgeon to keep in storage pieces of skin, periosteum, bone, cartilage, blood vessels, peritoneum, omentum and fat, ready to be used." (Dr. Alexis Carrel, 1912.)

Simplified Herniorrhaphy in Infants and Young Children

WILLIAM HOFFMAN, M.D., *Brooklyn, N. Y.*

REPAIR of inguinal hernia in infants and young children can be performed without disturbing the external inguinal ring. The operation is based on the generally accepted theory that a hernia in this age group is cured just by high ligation of the sac. The procedure is simple and satisfactory and deserves greater emphasis.

In the usual herniorrhaphy, the external oblique aponeurosis is incised down to and



FIGS. 1–7. Steps in the simplified repair of hernia. Note that the external ring is intact throughout.

including the external ring before proceeding with the repair. Adequate ligation of the hernial sac is performed without difficulty, but one frequently has to reapproximate a frayed or tenuous external oblique aponeurosis. In addition, there is always the problem of how tightly to close the external ring. Occasionally one is too zealous and the result is a swollen testis and possibly a non-fertile organ in later years.

The following method avoids these difficulties (Figs. 1–7 are self-explanatory): A small transverse incision beginning ½ inch above the pubic tubercle and extending laterally for about 1½ inches is made and deepened to expose the external oblique aponeurosis. The aponeurosis is incised in the line of its fibers beginning ¼ inch from the external ring and extending laterally a distance of 1 inch. *The external ring itself is not incised or changed in any way.* The edges of the incision in the aponeurosis are gripped with tiny hemostats and separated, uncovering the spermatic cord in the inguinal canal at or near the internal ring. The cremasteric fascia is incised and the sac which is invariably of the indirect variety and thus lies anteromedially in relation to the cord structures is identified, dissected free, and high ligation performed in the usual manner. The excess sac is excised and the opening in the cremasteric fascia closed with two or three interrupted sutures. The edges of the external oblique aponeurosis are reapproximated in similar fashion. The skin is closed with a continuous subcuticular stitch of very fine catgut to avoid later removal of sutures. I prefer to use fine black silk for the repair itself.

It will be noted that at no time in this procedure has the external ring been touched or otherwise displaced. As a matter of fact, except for dissection of the hernial sac the spermatic cord itself has not been disturbed. This procedure appears to give a maximum of exposure with a minimum of trauma. It is particularly suited for the usual inguinal hernia in infants and young children. When complications such as strangulation or inguinal wall defects are present, the surgeon may resort to the standard hernia operation.

An Appraisal of Temporary Gastrostomies and Enterostomies

CHARLES B. RIPSTEIN, M.D. AND KEITH M. SCHNEIDER, M.D., *New York, New York*

From the Department of Surgery, Albert Einstein College of Medicine and the Surgical Service of the Bronx Municipal Hospital Center, Bronx, New York.

GASTROINTESTINAL decompression is a vital adjunct in surgical treatment. However, nasogastric or small intestinal intubation may have significant deleterious effects. At best, it is distinctly distressing to the patient; at worst, it can lead to serious complications and even to death [1].

Major complications of intubation fall into five major groups. These include respiratory problems, laryngeal obstruction [2], esophageal ulceration and eventual stricture, perforation of the stomach or small intestine, and small intestinal obstruction [3]. Other complications are ulceration of the nasal septum, otitis media and tubotympanitis. Many of the untoward effects of intubation are late in development and frequently the operating surgeon is completely unaware of them. Also, it should be emphasized that while the majority of these problems arise from prolonged use of these tubes, important sequelae can and do follow one or two days of intubation.

We have been convinced therefore that alternative measures such as gastrostomy or jejunostomy would reap rich dividends in the avoidance of many postoperative pitfalls. Since the use of these technics was started, numerous other advantages have come to light.

INDICATIONS

Our indications for the performance of either gastrostomy or jejunostomy are rather simple. It is believed that any patient who may require prolonged nasogastric suction is a candidate for a gastrostomy. This is especially true in the aged and when the patient manifests some evidence of respiratory disease such as marked chronic bronchitis or bronchiectasis. Furthermore, this method is used if we expect difficulty in postoperative feeding, as is not uncommon in debilitated patients with carcinoma. Finally, we use gastrostomy as a vent in procedures performed on the esophagus (i.e., esophagogastrectomy). This prevents distention of the anastomotic line initially and later is available for feeding.

Jejunostomy is indicated in those cases in which open decompression of the small intestine is performed at laparotomy. It has been our policy in cases of small intestinal obstruction or marked paralytic ileus to enter the small intestine about 18 inches from the ligament of Treitz and deflate the bowel by means of the double-lumened Devine tube. (Fig. 1.) Following this a catheter jejunostomy is performed.

TECHNIC

A Stamm type of gastrostomy or jejunostomy is employed. Two concentric purse string sutures of No. 3-0 silk are placed in the bowel or stomach wall. After opening the viscus a No. 20 French catheter is inserted for a distance of 3 to 4 inches. A No. 3-0 chromic catgut suture is then passed through the wall of the catheter and sutured at the very edge of the incision in the bowel wall. (Fig. 2.) By pushing in on the tube and simultaneously tying the purse strings, very adequate inversion is achieved. No attempt is made to perform a

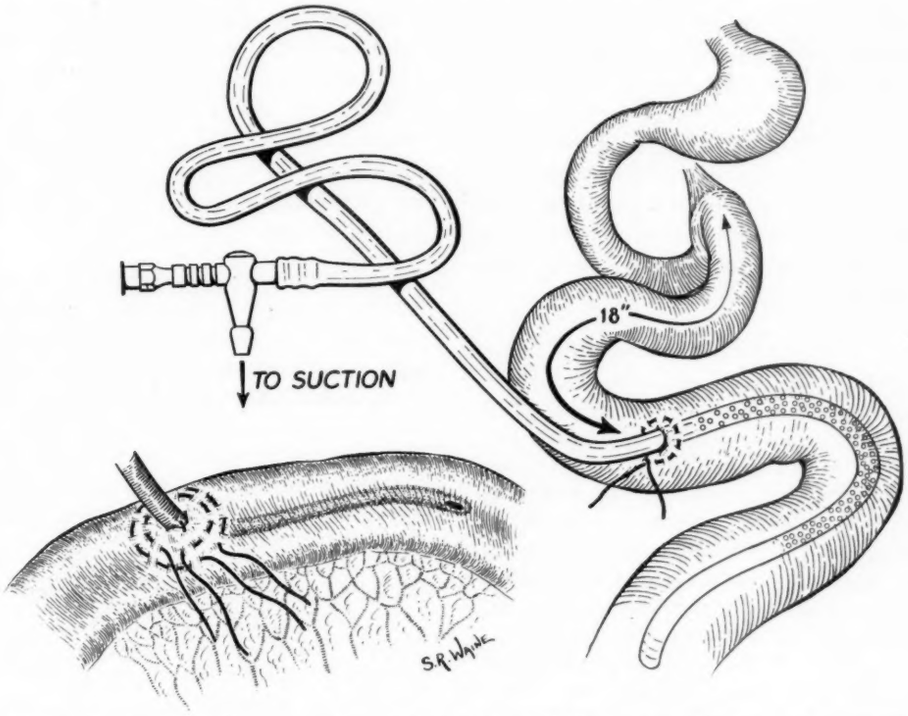


FIG. 1. Devine tube decompression 18 inches from the ligament of Treitz and insertion of catheter.

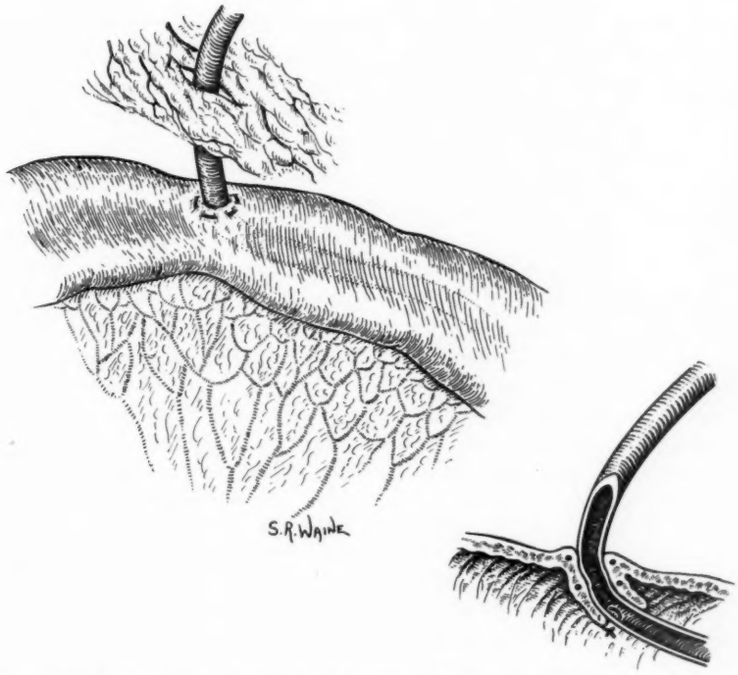


FIG. 2. Inversion of catheter in jejunum with two concentric purse string sutures. Note catheter being pulled through the greater omentum.

Witzel tunnel [4]. The catheter is then withdrawn through the greater omentum and brought out through a stab wound in the left flank.

We do not suture or even pull stomach or small intestine to the anterior abdominal wall, believing that this may act as a point around which twisting and obstruction can occur. No complications from our technic have been observed to date.

CASE REPORTS

CASE I. (*Pre-existing respiratory disease.*) C. P. (No. 9875), a seventy-two year old white man with chronic bronchiectasis and pulmonary emphysema, was operated upon for sigmoid diverticulitis one year prior to the present admission. Following the sigmoid resection his postoperative course was complicated by severe pneumonitis. A stormy convalescence ensued. He was re-admitted one year later with signs of an obstructing lesion at the old anastomotic site and laparotomy revealed a neoplasm at that point. A subtotal colectomy and tube gastrostomy were performed and the Levin tube was removed at the termination of the procedure. The gastrostomy was kept on suction for three days, after which it was clamped. Oral feedings were well tolerated and the tube was removed on the sixth postoperative day. No respiratory complications were encountered.

Comments. In view of the findings of bronchiectasis and pulmonary emphysema and because of the pneumonitis that followed the first operative procedure, it was thought that nasogastric suction was contraindicated. We have no doubt that the absence of the Levin tube allowed the patient to clear his secretions in a more effective manner and thus prevent respiratory problems.

CASE II. (*Postoperative alimentation.*) S. T. (No. 35033), a sixty-six year old white woman, was admitted in a markedly cachectic state with unremitting obstructive jaundice. At the time of perfoming a cholecystojejunostomy for inoperable carcinoma of the pancreas, a tube gastrostomy was performed. The patient refused all oral feedings for two weeks following the procedure. However, the gastrostomy allowed us to instill high calorie feedings which were extremely well tolerated. The patient slowly began to gain weight on this regimen, and at the end of the second week began to resume her oral intake. Shortly thereafter the gastrostomy tube was removed and the patient was discharged after the resolution of a moderately severe wound infection.

Comments. In dealing with debilitated patients with advanced neoplasms it certainly is not uncommon to find the patient refusing feedings in the postoperative period. Intravenous fluids are only a poor and incomplete substitute. Tube gastrostomy, by allowing early and adequate feeding, can in this situation be life saving.

CASE III. (*Decompression of anastomosis.*) M. B. (No. 81019), a fifty-two year old white man, was admitted with a neoplasm of the stomach involving the cardia and extending into the esophagus. The surgical procedure consisted of an esophagogastrectomy and splenectomy. In addition to a Heinecke-Mikulicz pyloroplasty, a tube gastrostomy was inserted into the stomach remnant and brought out through a small stab wound in the flank. No Levin tube was used.

After four days small gastrostomy feedings were begun and on the tenth postoperative day oral intake was started.

After it was obvious that no leak had occurred, the tube gastrostomy was withdrawn and the wound was closed within forty-eight hours. The remainder of the postoperative course was without incident.

Comments. The performance of a gastrostomy has numerous advantages in esophageal gastric surgery. Primarily it allows excellent decompression of the anastomosis without the obvious disadvantages of a nasogastric tube lying against the suture line. Furthermore, if disruption of the anastomosis does occur, the tube is available for feeding while the esophagus is kept at rest. Finally, in these patients who are so frequently debilitated, this technic makes complete ingestion of food possible much earlier in the postoperative period. This decompression, by virtue of the large size of the lumen and the shortness of the tube, is much more effective and less likely to become obstructed by gastrointestinal contents.

CASE IV. (*Intestinal obstruction.*) V. P. (No. 38314), a twenty-seven year old white woman, showed signs of intestinal obstruction one week after the perfomance of a left salpingo-oophorectomy for a rupture tubal pregnancy. At laparotomy an obstructed loop of ileum was found to be gangrenous, with tremendous dilatation of the proximal small intestine. The distended bowel was then decompressed about 18 inches from the ligament of Treitz by means of the Levin tube. After resection of the gangrenous bowel, continuity was restored by end-to-end anastomosis. A tube

jejunostomy was then inserted through this same incision.

The postoperative course in this patient, who was extremely ill at the time of surgery, was surprisingly benign. The jejunostomy drained about 500 cc. for the first three days and then the amount gradually decreased. The tube was removed on the tenth postoperative day.

Comments. For patients who at surgery are found to have markedly dilated bowel, either from obstruction or severe paralytic ileus, we believe that intestinal decompression is mandatory. It allows for the much quicker return of function of the bowel and "opening" of the anastomosis. In addition there is no question that distention impedes adequate ventilation. In infants and in old age this is frequently the forerunner of atelectasis and pneumonitis. Finally, by preventing decrease in the rate of venous flow secondary to distention, the incidence of venous thrombosis and pulmonary embolism should be diminished. The insertion of the temporary jejunostomy merely prevents the reaccumulation of intestinal gas as so often happens in bowel that has been made atonic by marked dilatation. If necessary, the jejunostomy also acts as a portal for postoperative alimentation.

CONCLUSIONS

Gastrostomies and jejunostomies are safe and effective methods of avoiding the complications of nasogastric intubation. In the aged and the very young they may make the difference between a successful or a fatal outcome. Other advantages include their use in postoperative alimentation and as a vent for the protection of upper gastrointestinal anastomosis.

Another striking benefit derived from these procedures is in the postoperative comfort of the patients. Their morale is higher and, in general, they are much less apprehensive and more cooperative. This method of gastrointestinal intubation has been used in fifty cases with no untoward effects.

SUMMARY

The use of tube gastrostomies and jejunostomies is encouraged. Case reports are presented which show clearly the advantages of this technic over nasogastric or small intestinal intubation in the immediate postoperative period. With the use of these methods the late sequelae of indwelling nasal tubes are also completely avoided.

REFERENCES

1. Farris, J. M. and Smith, G. K. An evaluation of temporary gastrostomy. *Ann. Surg.*, 144: 475, 1956.
2. Iglauer, S. and Molt, W. F. Severe injury to the larynx resulting from indwelling duodenal tube (case reports). *Ann. Otol., Rhin. & Laryng.*, 48: 886, 1939.
3. Eade, G. G., Metheny, D. and Lundmark, V. O. An evaluation of the practice of routine postoperative nasogastric suction. *Surg., Gynec. & Obst.*, 101: 275, 1955.
4. Bisgard, J. D. Gastrostomy-jejunal intubation. *Surg., Gynec. & Obst.*, 74: 239, 1942.

A Simplified Technic of Operative Cholangiography

GILBERT L. WRIGHT, M.D., *Salt Lake City, Utah*

From the University of Utah Medical School, Salt Lake City, Utah.

THE apparatus used in this technic consists simply of a 20 cc. syringe with an attached No. 18 or No. 20 needle and a 12 to 18 inch length of polyethylene tubing which fits the needle snuggly, plus a No. 18 or No. 20 needle with the hub and the point cut off leaving a cylinder with two blunt ends. (Fig. 1.) The No. 18 needles are ordinarily used except in cases in which the cystic duct is unusually small and will admit only the No. 20 needle shaft.

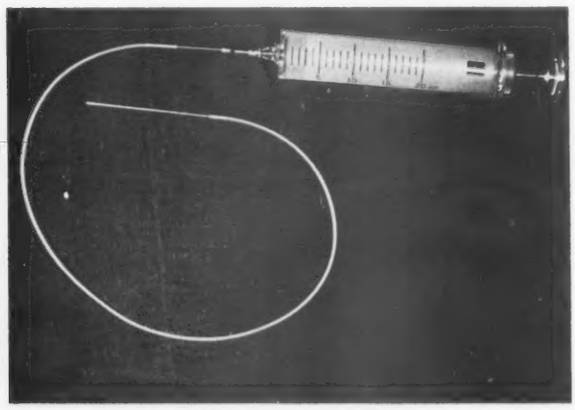


FIG. 1. Simple apparatus used in operative cholangiography.

The cystic duct is exposed and identified, and a small incision is made in its distal portion, well away from its junction with the common bile duct. The needle is inserted and held by a right angle clamp placed on the duct and contained needle. A ligature is then applied to hold the needle securely in the duct and the clamp is removed. If difficulty is encountered in passing either the No. 18 or the No. 20 needle, the gallbladder can be punctured at its junction with the cystic duct by the needle cylinder, and a clamp applied and the dye injected at this site. If the gallbladder has been previously removed, the needle cylinder can be inserted directly into the common bile duct. The dye and tubing are filled with dye (35 per cent diodrast®) before inserting the blunt needle in the duct. This is to eliminate air bubbles which are easily confused with calculi. Five to 7 cubic centimeters is injected, the anesthetist arrests the patient's breathing momentarily and the x-ray is made. The film is inserted in a cassette tunnel placed beneath the patient before the start of the operation. Ordinarily a scout film is taken when the cassette tunnel is first positioned to make sure that the position and exposure are satisfactory. After the first film is exposed, it is removed and a second film is inserted. The remainder of the dye from the 20 cc. ampule is injected and a second exposure is made. While these films are being developed in a darkroom adjoining the operating corridor, a matter of four to five minutes, the gallbladder is dissected off the liver and removed, and the gallbladder bed is sutured. The films are then examined in an x-ray view box in the operating room and, if normal, the needle and tubing are removed, the cystic duct is ligated close to its junction with the common bile duct, the distal portion is amputated and the abdomen is closed. The whole procedure, skin-to-skin, takes about one and a half hours unless the duct has to be opened to remove stones or other obstruction. Cholangiography should not add more than ten minutes to cholecystectomy. A small quantity of dye (5 to 7 cc.) is used for the first injection in order to avoid too great a density of the opaque medium in the duct, as this may obscure small stones. A larger quantity is used for the second injection in order to fill the

biliary tree and assist passage of the dye into the duodenum.

I have used various methods of operative cholangiography and found this the simplest, quickest and most satisfactory. When the polyethylene tubing without the needle is used, difficulty is often experienced inserting it into the duct because of its flexibility. With the rigid needle shaft, insertion is usually easy, even through a small duct and through the valves of Heister. A tight ligature may occlude the compressible tubing in the duct but cannot occlude the steel needle shaft, nor can a clamp be applied to the duct with the tubing in it, as it can to the duct containing the firm needle shaft.

Exchange transfusion needles, which are woven flexible needles, have been used but these are larger in diameter and too flexible. If the needle is attached directly to the syringe, the syringe will be inside the wound and difficult to control during injection and exposure, hence the polyethylene tubing.

I am convinced of the value of operative cholangiography, as described by numerous previous writers on the subject (notably Hicken et al.), and in a number of cases I have found calculi in the common bile duct which were not suspected by clinical signs and symptoms. It is particularly valuable when stones are present in the common bile duct and removed. A cholangiogram at this point will sometimes show stones still present in a duct which the surgeon had considered clear.

Soranus of Ephesus (2nd century, A.D.), a follower of the Methodist School of Aselepiades, was the leading authority on the gynecology, obstetrics and pediatrics of antiquity. His treatise on midwifery and diseases of women, preserved in Dietz's Greek text (Konigsberg, 1838), was the original of such famous works as Roslin's Rosengarten (1513) and Raynalde's Byrthe of Mankynde (1545). Most of the innovations in these books, such as the obstetric chair and podalic version, have been traced back to Soranus. After Soranus there were no real additions to the field of obstetrics before the time of Paré, some 1,500 years later. (T. S. W.)

"The loss of hair from syphilis (syphilitic alopecia) is of psychologic rather than pathologic interest, for there is a widespread delusion that syphilis commonly causes baldness—a delusion with no other foundation than the claim of quackery and the fact that syphilis is habitually contracted at the time of life when senile baldness begins—the age of twenty-five, alas! As a matter of fact, there are two varieties of syphilitic alopecia, neither of any great importance." (From: BRYANT, J. D. and BUCK, A. H. American Practice of Surgery. Baltimore, 1910. Williams Wood & Co., division of Williams & Wilkins Co.)

The Intraperitoneal Landmarks in McBurney Appendectomy

JAMES R. WATSON, M.D., *Pittsburgh, Pennsylvania*

THE gridiron incision is widely accepted as the preferable incision for the removal of an inflamed appendix, and there are many references in the medical literature which attest to the reduced mortality and morbidity attending its use. Furthermore, the incidence of incisional hernia is practically nil unless the incision is left open for the insertion of drains. The incision is usually adequate for its needs, and when this is not the case it can be enlarged by Weir's method in which it is extended medially with division of the fibers of the rectus sheath and medial retraction of that muscle. Knowledge of the intraperitoneal landmarks which indicate the location of the appendix eliminates the necessity of enlarging the incision in most instances, and in the average case permits its location without undue trauma and without unnecessarily disturbing the other structures in the right lower abdominal quadrant. The landmarks to be described are valid when used either with the standard McBurney incision or with one of its modifications. Not all appendectomies are easy and considerable trauma can be inflicted through even large incisions if the surgeon is not familiar with the detailed anatomy of the area.

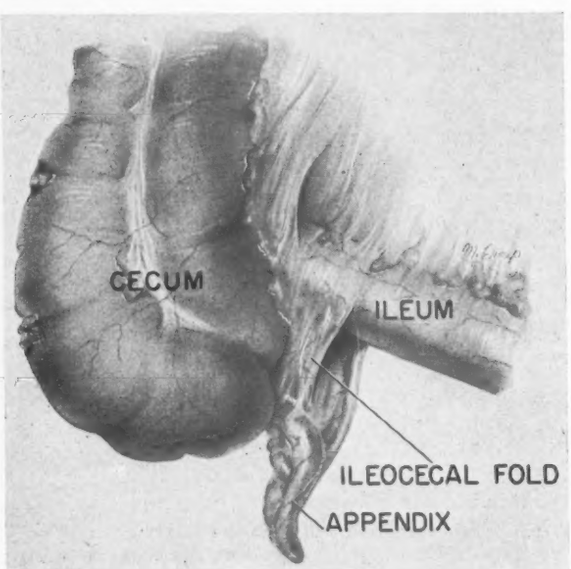


FIG. 1. The three intraperitoneal landmarks which indicate the location of the appendix.

There are three landmarks which are invariably present, any two of which will be adequate to reveal at least the base of the appendix. (Fig. 1.) Two of these have been described before. The third was first demonstrated to me by my father, the late Dr. Charles M. Watson, and I know of no article on the technic of appendectomy in which it is mentioned.

The *cecum* is the most prominent landmark and, when seen in the depth of the wound, can be grasped gently with soft, toothless thumb forceps and displaced upward until its base is located. This is the landmark most frequently used, for its size and location cause it to appear prominently in the depth of the wound in most cases. It is not the most reliable in that considerable manipulation may be necessary to reveal the appendix despite the fact that the anterior longitudinal band leads directly to its attachment with the cecum. It may be fixed to the posterior abdominal wall so that it cannot be brought up into the incision with ease. It may also be confused with a low lying transverse colon, although the attachment of the omental apron to the latter should eliminate this error.

The second landmark is the *terminal ileum* which can be distinguished from the remainder of the small intestine by the presence of longitudinal blood vessels which course along its antimesenteric side. These vessels are the anastomoses between the ileocolic and right colic arteries, are invariably present and are

found only on the terminal 2 inches of the ileum. The circulation to the remainder of the small intestine is through circular branches which arise from the mesentery. Thus, if a segment of small intestine is seen with longitudinal vessels coursing along its antimesenteric side, the surgeon may be certain that he is within 2 inches of the base of the appendix, and he can find it by gently displacing the terminal ileum to the patient's left.

The third landmark is the *ileocecal fold*, the so-called bloodless fold of Treves. It is a triangular fold of peritoneum, the upper border of which is fixed to the terminal ileum opposite its mesenteric attachment, while the lower free border, passing over the ileocecal junction, joins the mesenteriolum of the appendix and sometimes the appendix itself. Its counterpart on the upper side is the ileocolic (superior ileocolic) fold, and together they serve to brace the ileum in its end-to-side insertion into the cecum. The ileocecal fold is the more prominent of the two, varies considerably in size although it is usually quite distinct, and represents as reliable a guide as the longitudinal blood vessels of the terminal ileum. It falls like a hood over the base of the appendix, and when it is large, may completely conceal it. If it is lifted up and the ileum and cecum spread apart, the appendix, or at least its base, will appear. The maneuver is of particular value when the appendix lies in a retrocecal position, for once the base is identified, its course can be determined readily and the proper procedure instituted for its removal.

"John of Arderne was the first English surgeon worthy to be classed with the men who were recreating surgery in Europe. He was born in A.D. 1307, while Lanfrank, the most distinguished pupil of William de Salicet, had died only in A.D. 1306. Henri de Mondeville had gained notoriety by his refutal of the doctrine of healing by second intention, and Guy de Chauliac had yet to achieve his title of Prince of Surgeons. Like his distinguished European contemporaries, John of Arderne was trained in the wars. He seems to have served under two Dukes of Lancaster in succession, at Algeçiras, at Antwerp, and later in Aquitaine. After a long apprenticeship to military surgery in the Hundred Years' War, he went to Newark, then a great centre of trade and an occasional meeting-place for the English Parliament. There he practised surgery from A.D. 1349 to 1370, devoting much of his attention to the surgical cure of fistulae—openings through the skin made by accident or disease and forming abnormal communications with some internal organ. At the age of sixty-seven, when his practice was large and his reputation great, he came to London and joined the Guild of Military Surgeons. He knew well many of the noble and princely families of his time." (From: GRAHAM, H. Surgeons All, p. 118. New York, 1957. Philosophical Library, Inc.)

CASE REPORTS

Gastrojejunocolic Fistula with Associated Islet Cell Tumors of the Pancreas

KENNETH H. MACGREGOR, M.D., *Brooklyn, New York*

From the Department of Surgery of the Brooklyn Hospital Brooklyn, New York.

THE coexistence of peptic ulcer and endogenous hyperinsulinism is considered rare in spite of the fact that exogenous insulin can induce ulceration in the experimental animal [1].

Peptic ulcerations associated with islet cell tumors of the pancreas have been reported by Zollinger and Ellison [2]. Their cases showed marked gastric hypersecretion and hyperacidity. The islet cell tumors were of the non-insulin–producing variety. They postulate an ulcerogenic humoral factor of pancreatic islet origin and give reasons for implicating the hyperglycemic-glycogenolytic factor of the pancreas (glucagon). An electrophoretic analysis of the serum of one of their patients demonstrated a protein fraction with mobility characteristics similar to those of glucagon. It was not found in normal serum and elicited a hyperglycemic response in experimental animals.

CASE REPORT

C. M., a forty year old single woman, was admitted to the Medical Service of the Brooklyn Hospital on February 17, 1954. She complained of cramp-like abdominal pains, nausea and vomiting for a period of several months. There was an associated weight loss of thirty pounds.

This patient had first entered the Brooklyn Hospital in 1946 with an eight-year history of recurring attacks of fainting followed by unconsciousness and convulsions. The seizures occurred during periods of fasting. An admission blood sugar of forty-two mg. per cent was reported. Relief of symptoms was obtained by the administration of intravenous glucose. A diagnosis of insulinoma was made but no definitive therapy was instituted.

She was readmitted two years later because of hysteria and mental depression. A diagnosis of psychoneurosis, anxiety state, was made. The fasting blood sugar was seventy mg. per cent at this time.

In 1950, the patient was again admitted to the hospital because of a twenty-month history of epigastric pain not relieved by diet. A gastrointestinal series revealed a duodenal ulcer with marked deformity of the cap. A subtotal gastrectomy was performed for a duodenal ulcer penetrating into the pancreas. Palpation of the pancreas was normal.

She remained relatively well for several years, complaining of some weakness when abstaining from food or after exercise. However, the convulsive seizures had been infrequent since 1946. Beginning three months before the present admission in 1954, she noted recurring epigastric pains, loss of appetite, vomiting, and the previously noted weight loss of thirty pounds. The pain was not relieved by eating.

The physical examination revealed an emaciated woman in no acute distress. There was minimal epigastric tenderness.

The hemoglobin was 55 gm. per cent and the red blood cell count was about 3½ million per cu. mm.

The urinalysis was normal.

A gastrointestinal series showed a well functioning gastroenterostomy with about 50 per cent of the stomach remaining. There was no evidence of a marginal ulcer. A small pouch at the site of the anastomosis was noted, however.

Previous colon and genitourinary x-ray studies were negative.

Repeated fasting blood sugars were recorded.

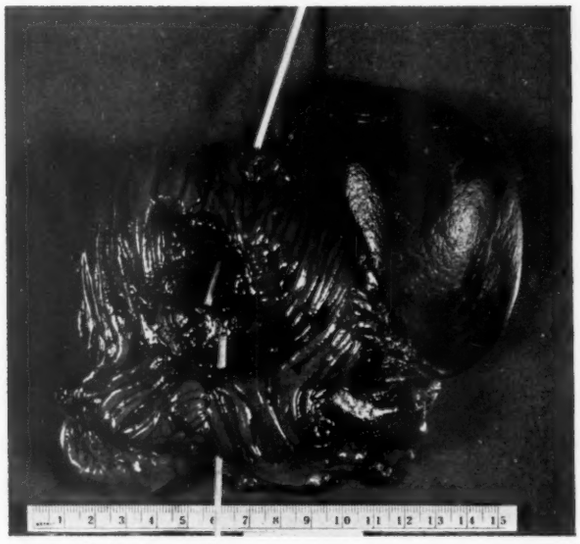


FIG. 1. Photograph of gross specimen. The stomach has been opened. The large ulcer crater is visible with pancreatic tissue seen in its floor. The lower pointer is directed from the colon and the upper pointer is directed from the jejunum, both communicating with the ulcer crater.

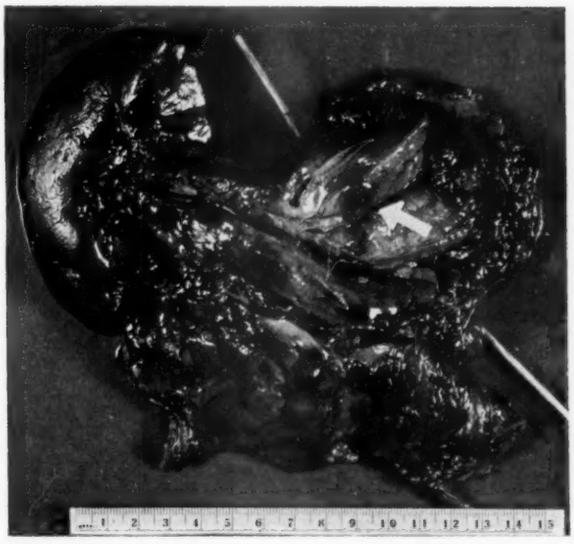


FIG. 2. Photograph of gross specimen from behind. The pancreas has been cut on its posterior surface. The probes pass from the colon and from the jejunum to the ulcer crater on the anterior surface of the pancreas. The arrow indicates the grossly visible islet cell tumor.

One was too low to read. Most of the others varied from 22 to 40 mg. per cent. Only one was over 50 mg. per cent.

The glucose tolerance test revealed a high sustained curve, reaching 340 mg. per cent at two hours and being 195 mg. per cent at three hours. The fasting report was 65 mg. per cent. The gastric analysis showed a fasting free hydrochloric acid of 12 units, with 54 units at thirty minutes and 34 units at sixty minutes.

The patient continued to vomit most of her food and her appetite remained poor. A surgical consultation was requested about two weeks after admission to determine the advisability of exploration for a probable islet cell tumor of the pancreas.

In view of the blurring of the diagnostic triad of hyperinsulinism by this patient's gastrointestinal complaints and loss of weight, it was thought that she might have a disorder of the gastroenterostomy stoma or possibly a malignant transformation as well as a functioning islet cell tumor. It was agreed to explore her with the thought that if the gastrointestinal findings were normal and if no tumor could be palpated in the pancreas, the body and tail would be removed as the most common sites of islet cell tumors, and then observe the clinical response of the patient, and await a complete pathological report on the tissue removed.

A subcostal chevron type of incision was made. Initial palpation in the left upper quadrant of the abdomen revealed an irregular mass approximately 8 cm. in diameter. It was very hard in consistency. After lysis of adhesions, this mass was found to consist of the antecolic gastrojejunostomy, the mid-portion of the transverse colon, and the body and tail of the pancreas. The head and neck of the pancreas palpated normally. The body of the pancreas seemed free posteriorly. At this time it was thought that we were dealing with a malignant neoplasm, possibly originating in an islet cell tumor.

The left kidney was not involved. The liver was normal to inspection and palpation. No enlarged lymph nodes were palpated.

It was decided to perform a block resection of the mass.* This was carried out in the following manner: a finger was inserted beneath the neck of the pancreas and while protecting the mesenteric vessels, the pancreas was transected between silk suture ligatures. The splenic vessels were then ligated along the upper border of the pancreas. The stomach was transected proximal to the anastomosis. The limbs of the jejunum were severed on either side of the gastroenterostomy. The transverse colon was next divided on both sides of the lesion. The specimen was removed with the spleen after freeing some posterior adhesions to the tail of the pancreas.

Gastrointestinal continuity was re-established by end-to-end anastomosis of the transverse colon and anastomosis of the jejunal limbs to the stump of the stomach in an antecolic position.

* The operation was performed in association with Dr. Harry Nicholas of the Surgical Residency Staff.

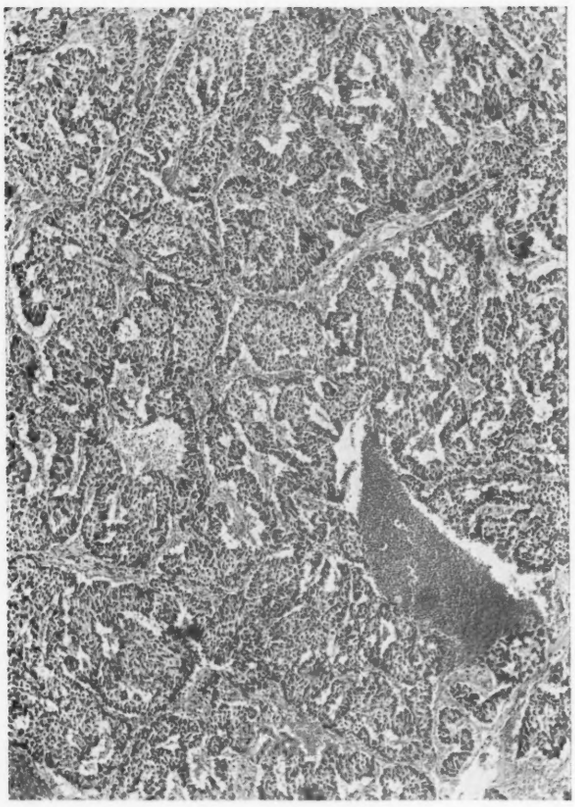


FIG. 3. Photomicrograph through the islet cell tumor. Irregular anastomosing cords with a resemblance to islet cell structure are seen. The cords are separated by a vascular loose connective tissue stroma. The cells are quite uniform and only occasional mitotic figures are seen.

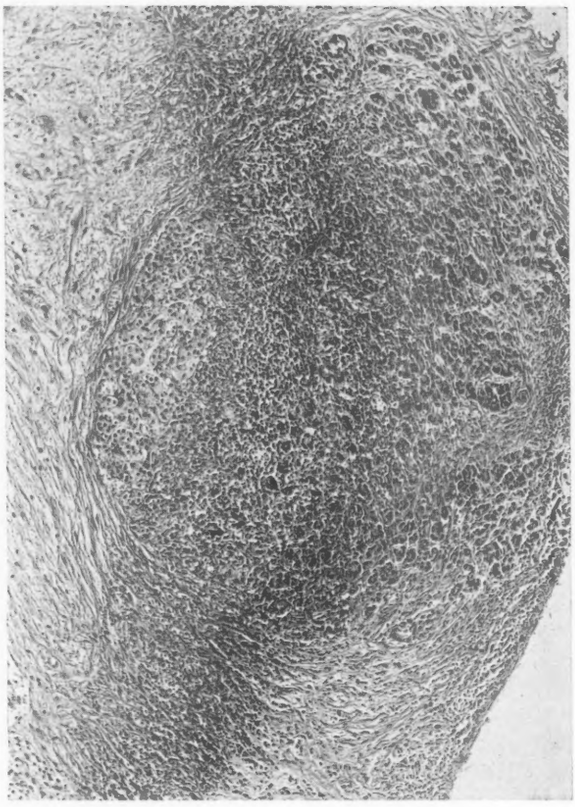


FIG. 4. Photomicrograph of the floor of the ulcer with inflamed pancreatic tissue bordering it. Some persisting pancreatic glands are seen in the inflamed tissue.

The pathological report was as follows: "The gross specimen is a 6.5 cm. cuff of gastric wall, a 10 cm. segment of jejunum, and a 13 cm. segment of colon, all three communicating with an ulcerated lesion measuring 4.5 cms. in its greatest dimension which erodes into one surface of a 10 cm. segment of pancreas. The spleen is attached to the specimen. (Fig. 1.) On sectioning the pancreas, a nodule, 1.5 cms. in dimensions, of pink, soft, glandular tissue appearing encapsulated and distinct from the remainder of the pancreas is noted. (Fig. 2.)

"The microscopic study of the pancreas shows a well circumscribed tumor composed of irregular anastomosing cords of eosinophilic cells with oval or round nuclei. These cords are separated by an extremely vascular loose connective tissue stroma. The cells are quite uniform and only occasional mitotic figures are seen. All of the tissue structures have a resemblance to islets of Langerhans'. (Fig. 3.)

"Nearby, but separate from the above described tumor, is a small nodule composed of more compact cellular structure in cords and small alveoli. The nuclei are round and the cytoplasm is eosinophilic. These cells closely resemble the acinar cells of the pancreas. Three additional adenomas of islet cell type are found.

"There is a large ulceration of the jejunum which has eroded into the pancreas. The microscopic study in this area shows the floor of the ulcer with inflamed pancreatic tissue bordering it. Some persisting pancreatic glands are seen." (Fig. 4.)

The final pathological diagnoses were: (1) Islet cell adenomas of the pancreas; (2) acinar cell tumor of the pancreas and; (3) chronic marginal ulcer (postgastrojejunostomy) with penetration into the pancreas and autoanastomosis with the transverse colon.

The early postoperative course was uneventful. While the fasting blood sugar was 33 mg. per cent two days before the operation, the blood sugar rose to 475 mg. per cent on the first day postoperatively. The patient was receiving glucose infusions, however.

One week later the fasting blood sugar was 123 mg. per cent and it remained at about this level during the subsequent weeks of observation.

The patient did well for about a year, gaining weight and having no fainting attacks. The gastrointestinal complaints were minimal.

After this, however, she became quite psychotic, refused to eat, rejected all laboratory tests and the suggestion to be hospitalized, and died of inanition some six months later. No autopsy was obtained.

COMMENTS

Review of this case suggests that hyperinsulinism was a factor in the ulcer production as it is in the experimental animal. However, in view of the clinical rarity of the association as recorded in the literature, it is possible that the multiple islet cell adenomas noted in the pathological examination were of the mixed variety, some functioning and some nonfunctioning. Thus, in addition to the hypoglycemic factor, another factor producing the ulcerogenic response, as suggested by Zollinger and Ellison, could have been present.

SUMMARY

A case report is presented demonstrating a gastrojejunocolic fistula associated with multiple islet cell tumors of the pancreas. Clinical behavior and laboratory studies indicate hyperinsulinism of an intermittent variety.

Mental deterioration is known to occur as a result of repeated attacks of hypoglycemia and it may become irreversible, a fact that seemed to contribute to the demise of this patient after an initial period of postoperative improvement.

REFERENCES

1. Janowitz, H. D. and Crohn, B. B. Hyperinsulinism and duodenal ulcer; a rare clinical combination. *Gastroenterology*, 17: 578–580, 1951.
2. Zollinger, R. M. and Ellison, E. H. Primary peptic ulcerations of the jejunum associated with islet cell tumors of the pancreas. *Ann. Surg.*, 142: 709–728, 1955.

Acute Myasthenia Gravis Following Surgery for Acute Dissecting Aortic Aneurysm under Nitrous Oxide, Pentothal-Curare Anesthesia

W. Dean Warren, M.D., Douglas W. Eastwood, M.D. and William H. Muller, Jr., M.D.,
Charlottesville, Virginia

From the Departments of Surgery and Anesthesiology, University of Virginia School of Medicine, Charlottesville, Virginia.

Persons with myasthenia gravis have a remarkable sensitivity to curare preparations. As early as 1901 Oppenheim [*13*] pointed out the marked resemblance between the symptoms of curarization and this disease. Harvey and Masland [*9*] and Johns et al. [*10*] have strengthened these clinical observations by demonstrating the close similarity of the electromyographic changes in myasthenia gravis and curarization. In a group of carefully studied patients, Bergh [*2*] found those with myasthenia gravis to be five times more sensitive to curare than the control. Although it is generally agreed that curare anesthesia is contraindicated in these patients, it seems inevitable that occasionally in a patient with latent myasthenia gravis a coincidental disease will develop requiring emergency surgical therapy. Under such circumstances the preoperative diagnosis of myasthenia gravis is exceedingly difficult.

In 1953 Van Nouhuys [*13*] reported a well documented case in which myasthenic crisis followed a surgical procedure in a patient not known to have the disease. The patient was not given curare but was noted to have a profound response to sodium pentothal,® and in the postoperative period overt signs of myasthenia gravis developed. In a recent symposium [*12*] an additional case was reported which followed the administration of 16 mg. of curare during a surgical procedure. Each of these patients survived the acute episode precipated by surgery. Foregger [*6*] reported twenty-two fatalities following the use of curare, but in none was an unequivocal diagnosis of myasthenia gravis established.

Acute myasthenic crisis led to the death of a patient admitted to the University of Virginia Hospital who had surgical correction of an acute dissecting aortic aneurysm.

CASE REPORT

The patient, a sixty-two year old white farmer, was seized with a sudden, excruciating substernal pain which was followed by loss of consciousness. He was admitted to a local hospital with a diagnosis of acute myocardial infarction. His condition did not improve during the ensuing five days and Dr. Julian R. Beckwith of the University of Virginia Hospital was called in consultation. Electrocardiographic examination was essentially normal, but a repeat roentgenogram of the chest revealed definite widening of the descending aorta when compared with one made earlier. A diagnosis of acute dissecting aneurysm of the aorta was made

The patient was transferred to the University of Virginia Hospital on June 1, 1957. Physical examination on arrival revealed that he was in moderately acute distress, but rational and cooperative. Blood pressure was 180/100 mm. Hg, and pulses were normal and equal bilaterally in both upper and lower extremities. The heart was slightly enlarged with heart sounds of good quality; a systolic murmur was present at the apex and an early diastolic murmur was noted in the aortic area. The remainder of the physical examination was essentially non-contributory. Laboratory data revealed that the urine, concentrated, was free of red blood cells and albumin; blood urea was normal, and there was no evidence of hematological abnormality.

The patient was taken immediately to the operating room, anesthetized with sodium pen-

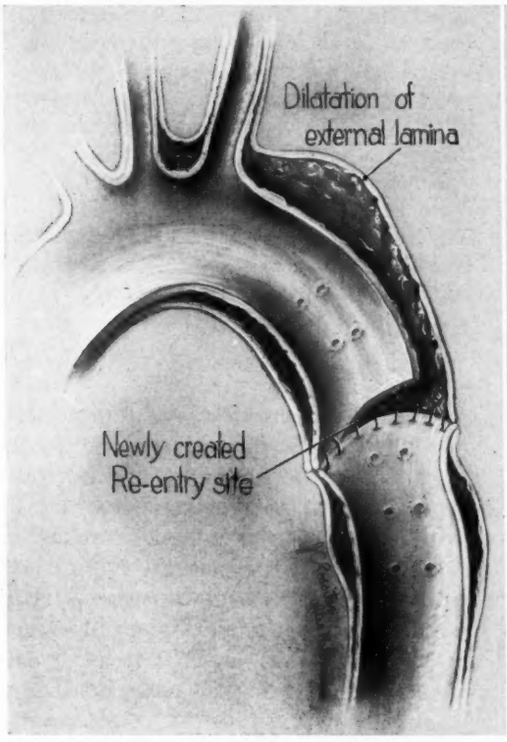


FIG. 1. Completed fenestration procedure.

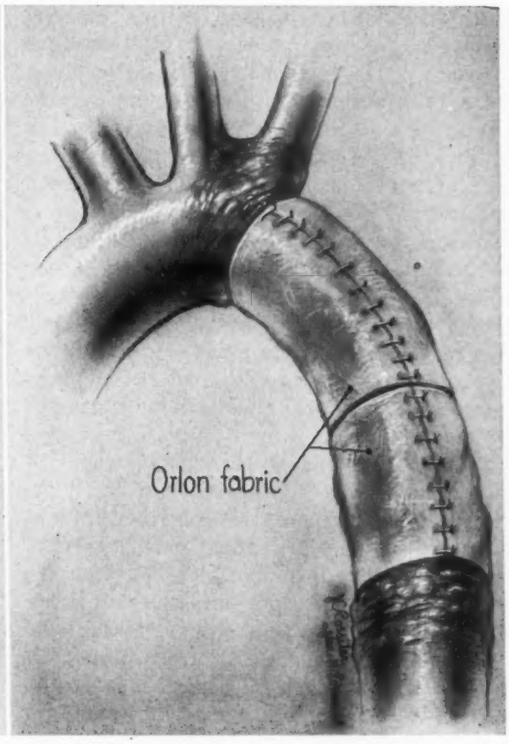


FIG. 2. Method of external support.

tothal, nitrous oxide and curare anesthesia, and an endotracheal tube was inserted. Hypothermia was utilized with the body temperature lowered to about 34°C. (rectally), and a trans-sternal incision was made through the fourth intercostal spaces. The dissecting aneurysm originated in the transverse and descending portion of the arch of the aorta and extended to the diaphragm; there was no intrapericardial involvement. The fenestration technic, as described by DeBakey et al. [5], was utilized with excision of an intimal window and closure of the opening into the distal aneurysmal channel. (Fig. 1.) After the re-entry site had been created and the anastomosis completed, the aorta just distal to the left subclavian artery was wrapped with orlon fabric for additional external support. There was a definite bulge in this area and the wall appeared to be quite thin. A second piece of orlon fabric was placed over the aortotomy to prevent further dilatation in this area. (Fig. 2.) Chest catheters inserted bilaterally were connected to water-sealed drainage. A rise in blood pressure to systolic levels of 220 mm. Hg was controlled with intravenous arfonad® and intermittent inferior vena caval occlusion. In conjunction with nitrous oxide and oxygen, 175 mg. of sodium pentothal and 27 mg. of curare were administered during the operative procedure. A continuous administration of norepinephrine was started to correct a moderate hypotension that had followed release of the aortic clamps.

Four hours after the conclusion of the operation respiratory motion was grossly inadequate. A tentative diagnosis of myasthenia gravis was made by the anesthesiologist (D. E.). About six hours following operation, with the patient fully cooperative, examination revealed a loss of lateral gaze bilaterally, bilateral ptosis, inability to raise the head and neck, and definite weakness of the extremities. His family was questioned and it was learned that unusual symptoms which had not been brought out in the history before operation had been present in years past. Periods of marked skeletal muscle weakness had prevented him from doing routine farm work. There had been difficulty in swallowing, definite periods of double vision and ptosis usually coming on at the end of the day. Although he had been studied at two large medical centers, a definite diagnosis had never been established and thyrotoxicosis, although not proved, had been suggested to account for his symptoms. During the five years prior to this admission, however, absence of these symptoms had enabled him to work without the aid of medication.

On the morning following operation, a neurological consultant (Dr. T. R. Johns) confirmed the diagnosis of acute myasthenic crisis precipitated by anesthesia and surgery. Cholinergic medication

resulted in return of lateral gaze, ability to speak more clearly, loss of ptosis, and improved strength and movement of the extremities. The patient was placed in a tank respirator; however, he became mildly hypoxic and fought the apparatus in an effort to breathe independently. He because disoriented, lost consciousness and an endotracheal tube was inserted and intermittent positive pressure breathing was begun. He was then removed from the respirator and a mechanical ventilator provided satisfactory aeration. Because his condition did not improve significantly, tracheostomy was performed to facilitate management of the airway and removal of secretions. Increased pressure was required to assure adequate ventilation.

On the night following operation restraints were needed to prevent displacement of the tracheostomy tube and other supportive devices. The norepinephrine requirements to control hypotension increased to 16 cc. of levophed® per 1,000 cc. of intravenous fluids at a rate of 100 cc. per hour. In addition, hydrocortisone and potassium chloride therapy was begun. The neostigmine dosage was maintained at 1.5 mg. every sixty to ninety minutes, utilizing the function of lateral gaze as the criterion for exact time of dosage. The potassium chloride was added to infusions to maintain normal blood potassium levels. The patient began to improve and on the fourth postoperative day norepinephrine was discontinued and aramine® was substituted. On the sixth postoperative day vasopressor agents were discontinued and he was able to tolerate periods up to thirty minutes without the aid of the respirator. Despite high humidity in the inhaled atmosphere, bronchial secretions became an increasingly difficult problem and bronchoscopy on the sixth postoperative day revealed purulent material originating from the left lower lobe.

On the tenth postoperative day a separation of the sternum and superficial layers of the transsternal wound was found and closed under local anesthesia without apparent difficulty. The following day there were longer periods of disorientation; difficulty in maintaining adequate respiration was encountered necessitating intermittent inflation of the cuff of the endotracheal tube. Neostigmine dosage was increased but muscle weakness invariably returned. Adequate ventilation was increasingly difficult. Fever, which had not been present for several days, occurred (104°F. rectally). In spite of increased antibiotic dosage and meticulous attention to removal of tracheobronchial secretions, the patient died on the fourteenth postoperative day.

At postmortem examination the most significant finding was a purulent tracheobronchitis and pneumonitis. There was an area of superficial necrosis on the tracheal wall. The area of the dissecting aneurysm was apparently stabilized as there had been no further bleeding and no proximal dissection. There was no indication of obstruction to the renal arteries or to other significant vessels. The intimal window was patent and there was no dilatation of the aorta in the region of the orlon fabric. It was concluded that the dissecting aneurysm had been satisfactorily controlled. The inability to initiate satisfactory spontaneous respirations in the face of an acute myasthenic crisis was the principal factor leading to death.

COMMENTS

Myasthenia gravis may be recognized by its clinical features in most instances. Many patients known to have this disease have undergone surgery successfully. In such circumstances adequate preparations can be made and the majority are brought through safely. Although curare anesthesia is contraindicated in these patients, it should be emphasized that an acute myasthenic crisis can follow a surgical procedure with any anesthetic. Indeed, Van Nouhuys [*13*] observed that his patient who had been anesthetized with sodium pentothal behaved as though curare had been used.

The initial problem presented by a patient such as ours is that of the differential diagnosis between prolonged curarization in a non-myasthenic and a true myasthenic crisis. A detailed history regarding the patient's past health should be obtained and, as was true in this case, may have to come from the family. One should seek out carefully any history of previous skeletal muscle weakness, diplopia, dysphagia, ptosis and other signs of myasthenia. A useful adjunct in establishing a diagnosis of myasthenia gravis is that of a therapeutic trial of drugs known to be of benefit in this condition. Although neostigmine is the time-honored medication for this disease, perhaps a more efficacious diagnostic tool is tensilon.® The desirability of this agent rather than neostigmine rests in its shorter duration of action, for occasionally a depolarizing agent intensifies the block rather than relieves it. Marked improvement of muscle weakness following administration of such a drug favors the diagnosis of myasthenia gravis. In this patient, shortly following the administration of neostigmine, there was a dramatic return of lateral gaze with disappearance of ptosis and immediate improvement in respiration. However, muscle weakness inevitably returned and was

hastened by exertion, such as a trial period without respiratory assistance.

Following the establishment of a correct diagnosis, there remain difficult problems in treatment. There are, in general, three aspects of management to consider: (1) pharmacological treatment for the deficit in neuromuscular transmission; (2) assurance of adequate ventilation; and (3) general supportive care.

Of these, the maintenance of satisfactory respiration is usually the initial problem to be faced, and this patient exhibited certain features which deserve special emphasis. The first of these is the necessity to maintain an adequate airway. Progressive muscle weakness of the pharynx and larynx may cause an upper respiratory obstruction similar to that seen in the unconscious patient. Some have advocated the use of an endotracheal airway or tracheostomy in the treatment of the myasthenic crisis, and in severe cases the routine use of tracheostomy and a respirator. A second type of respiratory obstruction more difficult to control is that related to inability to expel secretions from the lower respiratory tracts. The weakness of respiratory muscles makes it difficult or impossible for patients to cough and adequately expel secretions to a point at which endotracheal suction can be effective. Although extremely careful attention was given in this case to tracheobronchial aspiration, a completely satisfactory removal of secretions could not be accomplished and purulent tracheobronchitis with pneumonitis developed.

The maintenance of adequate respiratory exchange may be a severe problem in a man of great size and in this instance was compounded by the progressive pulmonary congestion. It was noted at the time of operation that considerable pressure was required to produce adequate respiratory exchange, and this continued through the postoperative course. With the onset of pneumonitis, difficult ventilation steadily increased and eventually an inflated endotracheal cuff was required which probably resulted in tracheal necrosis.

Pharmacological treatment rests essentially with the use of cholinergic drugs. Newer compounds have been promising but none have yet replaced the older methods of therapy. Neostigmine should be given in doses large enough to produce maximal amelioration of symptoms but, as has been emphasized recently [*8,12*], large doses of neostigmine may actually increase the muscle weakness. This has been called cholinergic crisis and may easily be confused with symptoms of myasthenia gravis itself.

Among the measures utilized for supportive care the most important is the use of antibiotics and high humidity to combat the danger of pulmonary infection. Progressive respiratory failure following the onset of pneumonitis is by far the most common sequence of events leading to death in these patients. Broad antibiotic coverage should be carried out and adequate dosage given as long as respiratory assistance is required. If neostigmine is required in large doses, atropine should be given to decrease tracheobronchial secretions and to lessen intestinal hypermotility. The importance of potassium ion therapy has been suggested and was utilized in this patient.

ACUTE DISSECTING ANEURYSM OF THE AORTA

An interesting facet of this case is that of acute dissecting aneurysm of the aorta. Only recently has the surgical approach to this highly lethal disease been undertaken. In 1955 DeBakey, Cooley and Creech [*5*] reported the first successful repair of an acute dissecting aneurysm of the aorta. Additional successful cases from this clinic [*14*] and elsewhere [*7,11*] have been published recently.

The decision to utilize hypothermia as an adjunct in the repair of the aneurysm was based upon our opinion that an attempt should be made to approach the primary intimal opening if the dissection involved only the ascending aorta. From a review of the cases in the literature it is abundantly clear that the establishment of a decompressing intimal window with closure of the opening into the distal aneurysmal sac does not necessarily prevent external rupture. In one of these cases reported from this clinic [*14*] as well as two others [*1,5*], rupture of the external wall of the aneurysm has been the cause of death. However, as the aneurysm involved only the distal arch and descending aorta, the plan to open the ascending aorta under hypothermia was abandoned. Experimental data [*3*] indicate that dissection following an intimal tear in the descending aorta extends little proximally, if at all, while distal progression is rapid. Consequently, the danger of intrapericardial rupture in this instance was extremely small. The possibility of definitive resection at this time was considered

but decided against because of the severity of the acute illness and the danger of impairing blood flow through the left common carotid artery. An area which has been a frequent site of external rupture of dissecting aneurysms is the region just distal to the left subclavian artery. Consequently, this area was wrapped snugly with orlon to prevent further dilatation, and additional supportive material was used to cover the region of the aortotomy. (Fig. 2.)

Autopsy revealed no evidence of rupture of the sac or extension of the dissection. The area of dissection appeared to be quite stabilized and could not be implicated in the patient's death. Deaths previously reported from this disease have been from complications directly related to the aneurysm such as intrapericardial rupture of the aorta or acute renal failure. Consequently, it is our conclusion that this should be considered a satisfactory repair of the dissecting aneurysm for the purpose of evaluation of fenestration procedures.

SUMMARY

Curare anesthesia is contraindicated in patients known to have myasthenia gravis. However, occasionally a surgical emergency arises and operation is performed on a patient not known to have the disease or with so-called latent myasthenia gravis. This was true of a case seen at the University of Virginia Hospital. Using nitrous oxide, sodium pentothal and curare anesthesia with hypothermia, surgical repair of an acute dissecting aortic aneurysm was performed on a sixty-two year old white man in whom a myasthenic crisis developed as a result of surgery and anesthesia. The patient died on the fourteenth postoperative day in spite of cholinergic medications and efforts to maintain adequate respiratory function. Postmortem examination revealed satisfactory repair of the acute dissecting aneurysm with death due to tracheobronchitis and pneumonitis.

The case history is presented and the general problem is discussed.

REFERENCES

1. Benson, W. R., Hamilton, J. E. and Claugus, C. E. Dissecting of aorta: report of a case treated by fenestration procedure. *Ann. Surg.*, 146: 111, 1957.
2. Bergh, N. P. Reaction of patients with myasthenia gravis to different agents causing neuromuscular block. *Scandinav. J. Clin. & Lab. Invest.*, 5 (Supp. 6): 11, 1953.
3. Blanton, F. S., Jr. Dissecting aneurysm—review and experimental study. Thesis submitted to the Graduate School of Medicine, Charlottesville, Virginia, in partial fulfillment of requirements for the degree of Master of Science in Surgery, June, 1957. Unpublished data.
4. Cooley, D. A. Discussion of paper by Warren, W. D. et al. [14].
5. DeBakey, M. E., Cooley, D. and Creech, O. Dissecting aneurysms of the aorta—surgical considerations. *Ann. Surg.*, 142: 586, 1955.
6. Foregger, R. Fatalities following curare. *J. A. M. A.*, 142: 1344; 1950.
7. Gilman, R. A. and Bailey, C. P. Surgical treatment of dissecting aneurysm. *J. Thoracic Surg.*, 33: 670, 1957.
8. Grob, D., Garlick, W. L., Merrill, G. G. and Freimuth, H. C. Death due to parathion: an anticholinesterase insecticide. *Ann. Int. Med.*, 31: 899, 1949.
9. Harvey, A. M. and Masland, R. L. A method for the study of neuromuscular transmission in human subjects. *Bull. Johns Hopkins Hosp.*, 68: 81, 1941.
10. Johns, R. J., Grob, D. and Harvey, A. M. Electromyographic changes in myasthenia gravis. *Am. J. Med.*, 19: 679, 1955.
11. Swann, W. K. and Bradsher, J. T., Jr. Acute dissecting aneurysm of the aorta: operation with recovery. *New England J. Med.*, 255: 36, 1956.
12. Tether, J. E. Management of myasthenic and cholinergic crises. *Am. J. Med.*, 19: 740, 1955.
13. Van Nouhuys, F. Myasthenia gravis and curare sensitivity. *Acta anaesth. belg.*, 3: 15, 1952.
14. Warren, W. D., Beckwith, J. and Muller, W. H., Jr. Problems in the surgical management of acute dissecting aneurysm of the aorta. *Ann. Surg.*, 144: 530, 1956.

Paraesophageal Bronchiogenic Cyst

ERNEST E. SCHNOOR, M.D., *Redwood City, California,* AND JOHN E. CONNOLLY, M.D., *San Francisco, California*

From The Department of Surgery, Stanford University School of Medicine, San Francisco, California.

WITH the increased frequency of routine roentgenograms of the chest, many asymptomatic mediastinal masses are being discovered. A small group of these densities are found to be bronchiogenic cysts. However, when a mass is found indenting the esophagus the diagnosis of bronchiogenic cyst is rarely considered. Smooth, circumscribed, intramural indentation of the esophageal lumen without perforation of the mucosa is usually caused by benign tumors of the esophagus such as leiomyomas or duplication cysts with a histological appearance of the normal esophagus or of the gastrointestinal tract.

We wish to report a case of intramural bronchiogenic cyst of the esophagus, and in so doing stress this entity in the differential diagnosis of intramural esophageal masses.

CASE REPORT

B. L. (SC No. B41213), a thirteen year old Negro girl, was first seen at Stanford University Hospitals on December 13, 1954, with complaints of labored breathing and pain between the shoulder blades of about one month's duration. Rhinitis and an acute localized gingivitis were the only abnormal findings on physical examination. A roentgenogram of the chest, however, revealed a faint density to the left of the spine behind the cardiac shadow. (Fig. 1.) The mass could not be identified on the lateral view.

The patient did not return for re-examination until September 25, 1956, at which time she complained of mild, recurrent "asthmatic attacks" in addition to her previously recorded symptoms. Examination again showed rhinitis with no other obvious abnormalities, but repeat roentgen studies with fluoroscopy of the thorax confirmed the presence of the mediastinal density noted previously.

An esophagram showed a smooth indentation of the esophageal lumen in the distal third on the left corresponding to the density noted on the plain films. (Fig. 2.) Esophagoscopy disclosed an intact mucous membrane with a narrowing of the lumen compatible with extrinsic pressure on the esophagus. No intrinsic lesion of the tracheobronchial tree was seen on bronchoscopy.

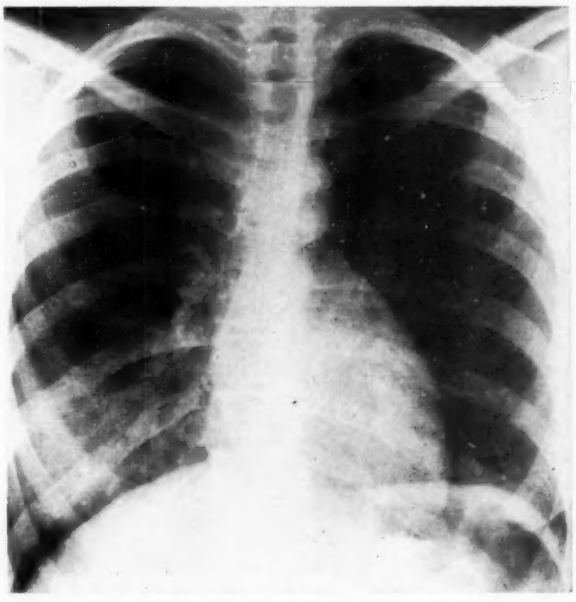
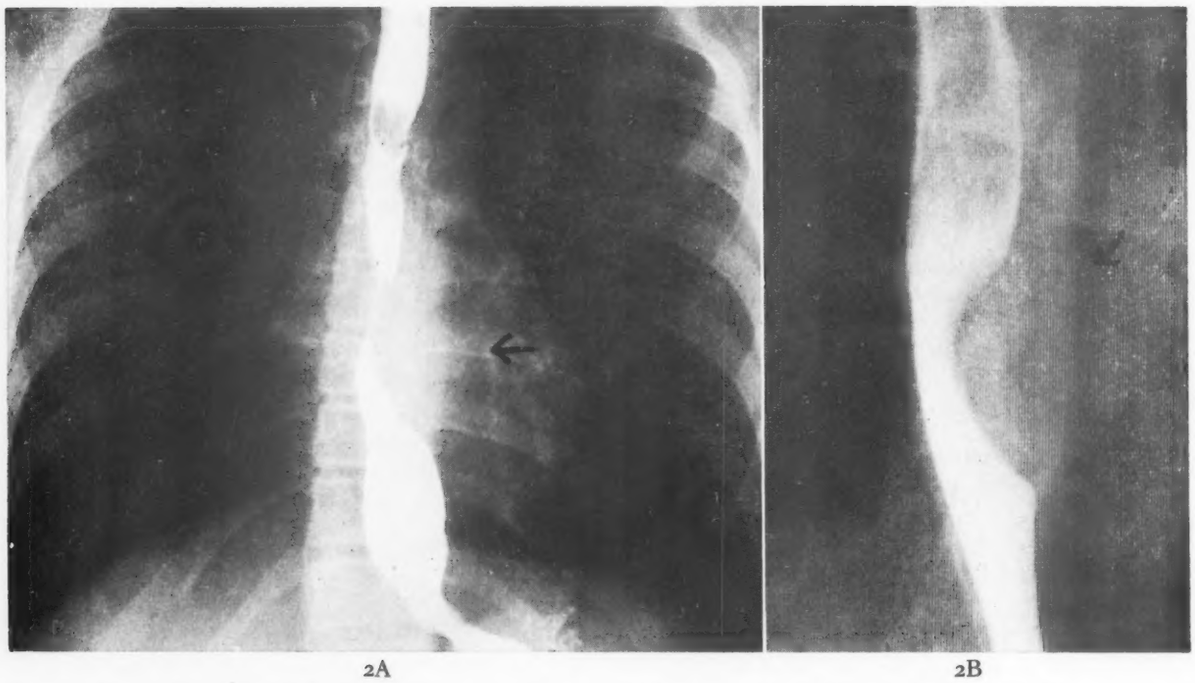


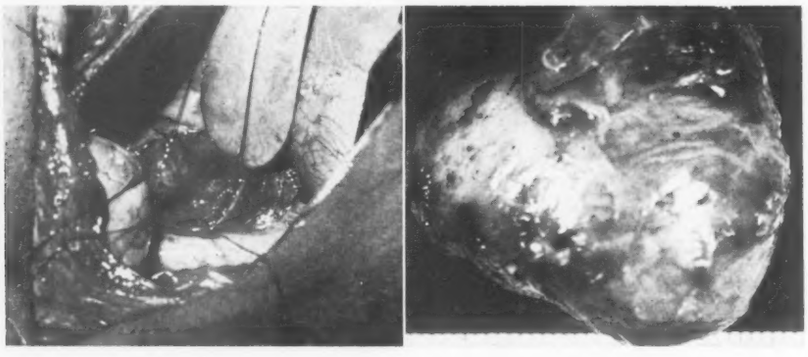
FIG. 1. Chest film showing faint density over dorsal spine behind heart.

On May 1, 1957, exploratory thoracotomy was performed under general endotracheal anesthesia through the left fifth intercostal space. In the posterior mediastinum there was a heart-shaped, firm, cystic structure, measuring 3.5 cm. in its greatest diameter, which was closely associated with the esophagus just caudal to the left inferior pulmonary vein. The pleura overlying the mass was incised in a longitudinal direction and the mass was mobilized by sharp and blunt dissection, carefully preserving the pulmonary vein and vagus nerve. (Fig. 3.) The cyst wall was found to incorporate fibers of the longitudinal muscle of the esophagus but did not involve the circular fibers. The postoperative course was uneventful and the patient was discharged from the hospital on the



2A 2B

FIG. 2. A, esophagram showing indentation corresponding to density seen in Figure 1. B, close-up of esophagram.



3 4

FIG. 3. Operative view of intramural cystic lesion.

FIG. 4. Gross appearance of removed specimen.

ninth postoperative day. Her subsequent course has been asymptomatic, without any "asthmatic attacks" to date.

The pathologic report revealed that the specimen consisted of a fluctuant mass measuring 3.5 by 3.0 by 2.5 cm. (Fig. 4.) It was a thick-walled cystic structure filled with brown viscous material. The wall was trabeculated and of fibrous consistency. Microscopic study (Fig. 5) revealed a three-layered muscular wall with a considerable amount of fibrous connective tissue surrounding foci of chronic granulation tissue and chronic inflammatory cells. One zone of hyaline cartilage was noted. The cyst lining varied in its structure. One area of pseudostratified ciliated columnar epithelium was found while another area resembled bowel mucosa but without goblet cells. A third zone revealed stratified squamous epithelium. The diagnosis was paraesophageal bronchiogenic cyst.

COMMENTS

True paraesophageal bronchiogenic cysts are extremely rare. We could find reports of only three such cysts in the literature, one each by von Westenryk [1], Robbins [2] and Maier [3]. There is an excellent review and classification

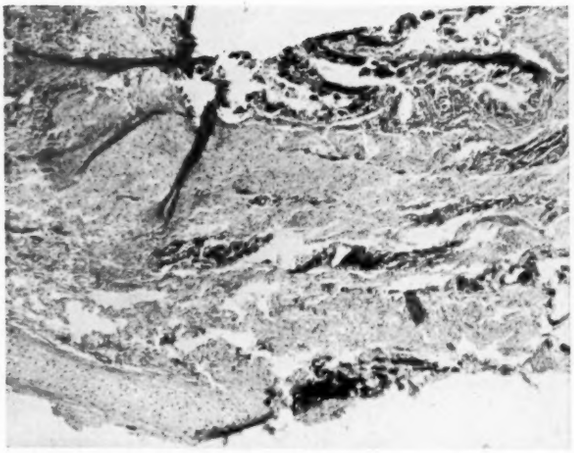


FIG. 5. Photomicrograph showing cartilage and columnar respiratory epithelium.

of these cysts by Laipply [4] and, more recently, by Maier [5]. These authors believe that the diagnosis of bronchiogenic cyst rests upon the findings of a cyst lined by ciliated columnar epithelium with a wall made up of smooth muscle and elastic connective tissue. In addition, the presence of cartilage is necessary for a diagnosis of bronchiogenic cyst.

The cyst reported herein showed not only the aforementioned characteristics but also contained tissue resembling bowel mucosa and stratified squamous epithelium. This would lend weight to the argument that such a tumor develops during the early embryonic stage of the host before the separation of the respiratory tract from the primitive foregut. The location of the cyst in the esophageal wall also lends support to this contention since bronchiogenic cysts have been found not only in close proximity to the trachea and bronchi but also in the pericardium [6,7], on the anterior aspect of the thoracic spine [8] and in the subcutaneous tissues near the sternal angle [9].

Cysts in the esophageal wall may cause serious tracheal compression and therefore should be removed even though they may be otherwise benign. Maier's [3] case of paraesophageal bronchogenic cyst produced chronic respiratory difficulty in a two year old boy. The cyst reported herein did not appear to encroach upon the tracheobronchial tree, although the patient had so-called "asthmatic attacks" which have not recurred since surgical excision of the cyst.

SUMMARY

A case of paraesophageal bronchiogenic cyst removed at surgery is reported. This is the fourth such case in the literature. The pathological diagnosis and theory of development are discussed.

REFERENCES

1. VON WESTENRYK. Zur Casuistik der Mediastinalcysten. *Prag. med. Wchnschr.*, 25: 373, 1900.
2. ROBBINS, L. L. The roentgenologic appearance of "bronchiogenic" cysts. *Am. J. Roentgenol.*, 50: 321, 1943.
3. MAIER, H. C. Tracheal compression from bronchogenic cyst in esophageal wall. *Am. J. Dis. Child.*, 80: 423, 1950.
4. LAIPPLY, T. C. Cysts and cystic tumors of the mediastinum. *Arch. Path.*, 39: 153, 1945.
5. MAIER, H. C. Bronchiogenic cysts of the mediastinum. *Ann. Surg.*, 127: 476, 1948.
6. JOEL, J. Teratom auf der Arteria pulmonalis innerhalb des Herzbeutels. *Virchows Arch. path. Anat.*, 122: 381, 1890.
7. RUSBY, N. and SELLORS, T. H. Congenital deficiency of the pericardium associated with a bronchogenic cyst. *Brit. J. Surg.*, 32: 357, 1945.
8. GUILLERY, H. Eine in die Wirbelsaule eigewachsene mediastinale Zyste (Vorderdarmzyste). *Zentralbl. allg. Path.*, 69: 49, 1937.
9. SEYBOLD, W. B. and CLAGETT, O. T. Presternal cysts. *J. Thoracic Surg.*, 14: 217, 1945.

Hemorrhage from Esophageal Varices—Its Prognostic Significance

EMILE L. MEINE, JR., M.D., *North Hollywood, California,* AND LEON J. WITKOWSKI, M.D., *Chicago, Illinois*

From the Fourth Surgical Service, St. Luke's Hospital, and Northwestern University Medical School, Chicago, Illinois.

ACUTE bleeding from esophageal varices is a serious and often fatal emergency which is not infrequently encountered. In one recent large series of patients with massive hemorrhage from the upper gastrointestinal tract, esophageal varices were the source of bleeding in 32 per cent of the cases [1]. There is frequently a tendency to treat this problem as conservatively as possible and, if the hemorrhage is controlled or subsides spontaneously, to place the patient on medical management and hope that he will not bleed again soon. This attitude is hardly tenable in view of reports which show that approximately 50 per cent of patients treated medically for portal hypertension die within one year after their first esophageal hemorrhage. Nachlas et al. [2], reporting on cirrhotic patients, found an initial mortality rate of 60 per cent with the first hemorrhage, while one-third of the survivors failed to live another year. Death results usually from exsanguinating hemorrhage or hepatic coma.

MATERIAL

The records of patients admitted to St. Luke's Hospital, Chicago, from 1950 through 1955 with acute bleeding from esophageal varices have been summarized in order to review their immediate course. During this six-year period there were forty-two admissions of thirty-three patients with acute esophageal bleeding. No cases of patients admitted for elective surgery or medical treatment with a previous history of esophageal hemorrhage but without recent bleeding were included. There were twenty males and thirteen females whose ages ranged from twelve to seventy-six years, with an average age of forty-eight years. The presenting symptom on admission was melena in thirteen instances and hematemesis in twenty-nine cases.

At the first admission to this hospital for hemorrhage, twenty-five of these thirty-three patients had a known history of liver disease, and twenty-three had bled within the previous two years. This was usually a lesser hemorrhage, and in five cases had been manifested by melena alone. Other significant findings on admission, as well as important facts from the past history, are listed in Table 1. Splenectomy had been performed on four patients previously. This usually makes a splenorenal anastomosis impossible and may make a direct portacaval shunt more difficult. Two patients had undergone multiple biliary tract operations, which would also render further surgical procedures more hazardous, particularly in the region of the portal vein. Alcoholism was listed only for those patients who admitted a moderate to heavy intake for a number of years. As to the type of underlying liver disease, portal cirrhosis was assumed to be present in twenty-nine cases, although this was confirmed in only a part of this group that underwent operation or autopsy. Although the majority were probably of Laennec's type, a few may have been posthepatitic in etiology. There were two cases of biliary cirrhosis following non-malignant obstruction of the extrahepatic biliary tract, and one definite case of postnecrotic cirrhosis in a man with prolonged exposure to carbon tetrachloride. There was only one proved case of extrahepatic portal vein obstruction.

PROCEDURES

The Sengstaken tube was used to control bleeding by esophageal tamponade in five cases,

and in two additional cases before transfer from other hospitals. Sixteen surgical procedures were then performed on fourteen patients. (Table II.) Even in this limited series a variety of procedures was performed by various members of the surgical staff, attesting to the lack of unanimity in treatment of this complex problem. A proposed splenorenal shunt was abandoned in one patient after an early cardiac arrest developed. This patient survived to leave the hospital. An attempt was made to reduce portal hypertension by some type of shunt procedure in ten of the fourteen patients, or to suture or resect the site of bleeding in most of the others. There was no surgical intervention in nineteen cases. There were five postoperative deaths and six deaths among non-operated patients, giving an over-all early mortality rate of 33.3 per cent. These data are summarized in Table III. Because this series is small and no separation of patients as to operative risk was made, no attempt has been made to correlate survival with operative or non-operative treatment. Although two-thirds of these patients had previously bled, it is apparent, as others have repeatedly reported, that the mortality rate is high and that acute bleeding from esophageal varices imposes a grave prognosis.

TABLE I

FINDINGS IN THIRTY-THREE PATIENTS ON FIRST ADMISSION TO ST. LUKE'S HOSPITAL WITH ACUTE BLEEDING FROM ESOPHAGEAL VARICES

Findings	No. of Patients
On physical examination:	
Hepatomegaly	22
Splenomegaly	17
Jaundice	11
Ascites	8
From the history:	
Alcoholism	12
Hepatitis	3
Previously known liver disease	25
Previous splenectomy	4
Multiple biliary tract operations	2

TABLE II

OPERATIONS PERFORMED ON FOURTEEN PATIENTS FOR VERY RECENT OR ACUTE BLEEDING FROM ESOPHAGEAL VARICES

Procedure	No. of Patients
Abdominal exploration, cholecystectomy	1
Exploration, early cardiac arrest	1
Direct portacaval anastomosis	2
Splenorenal shunt	8
Esophagectomy	1
Esophagotomy with suture of varices	1
Esophagogastrectomy	1
Esophagectomy and splenic artery ligation	1
Total number of procedures	16

TABLE III

TYPE OF THERAPY AND EARLY RESULTS IN 33 PATIENTS TREATED FOR ACUTE ESOPHAGEAL BLEEDING

Therapy	No. of Cases	No. of Deaths	Early Deaths (per cent of total cases)
Medical treatment only	19	6	18.2
Operative treatment of some type	14	5	15.1
Medical and surgical cases combined	33	11	33.3

CASE REPORTS

The following case reports are intended to show the complexity of the problem of acute esophageal bleeding in widely different age groups, as well as the difficult and sometimes frustrating surgical problems involved. These patients were operated upon by members of the general surgical and vascular surgical staff of St. Luke's Hospital (now Presbyterian-St. Luke's Hospital), Chicago.

CASE 1. K. W. (SLH 41014) was a young girl who had experienced her first hematemesis in 1946 at the age of six. One year later splenectomy had been performed. She bled again the following year, and in 1949 portacaval anastomosis was done. In the next several years she had four more episodes of hematemesis, two of which required hospitalization and blood transfusions. This twelve year old girl was admitted for the first time to St. Luke's Hospital on June 12, 1952, following a recent bleeding episode. Her hemoglobin was 9.0 gm. per cent, although bleeding, coagulation and prothrombin times were normal. There was 7.2 per cent bromsulphalein® retention at forty-five minutes. Other liver function tests were normal. Barium swallow demonstrated the presence of esophageal varices. Four days after admission,

splenorenal anastomosis was performed through a left thoracoabdominal incision. Right lower lobe atelectasis developed, but she quickly recovered and went home on the ninth postoperative day.

In March, 1953, this child had another large hematemesis. She was hospitalized locally, a Sengstaken tube was inserted and blood transfusions were given. Within a few days bleeding had apparently stopped and she went home. However, her stools continued to be tarry and she re-entered St. Luke's Hospital on March 19, 1953, eleven days after the most recent hematemesis. Her hemoglobin was 5.4 gm. per cent on admission. During the next five days she received 5,000 ml. of blood and the hemoglobin rose to 10.5 gm. per cent with a hematocrit of 37 per cent. With evidence of continued slow bleeding, she was again operated upon March 24, 1953, through a left thoracotomy incision. The esophagus was mobilized and the diaphragm opened. Large varices were found about the gastric cardia. The distal esophagus and upper third of the stomach were resected, and the stomach was drawn up into the chest and anastomosed to the esophagus. She received 3,000 ml. of blood during this operation. The patient had a low grade fever for five days postoperatively, but was fed on the eighth day and discharged on the thirteenth postoperative day. Her liver was not grossly nodular at either operation, and a liver biopsy specimen taken at the second procedure was essentially normal.

This patient was the youngest in this series, and the only one definitely shown to have extrahepatic portal obstruction. It is also surprising that splenorenal anastomosis was possible after an earlier splenectomy. Quite possibly this shunt subsequently closed, thereby predisposing to recurrent portal hypertension and bleeding which necessitated the last, more radical procedure.

Case II. J. K. (SLH 40106), a thirty-two year old, single, white man who operated a grocery, had two massive hematemeses at home. A history of his having a heavy alcoholic intake for the previous six years was obtained from his family. The patient was hospitalized locally and a Sengstaken tube was inserted. He continued to bleed and four days later, on February 28, 1953, he was transferred to St. Luke's Hospital. He was receiving his eleventh 500 ml. unit of blood on admission, which was noted to be type AB negative blood. The patient was disoriented and struggling wildly in restraints. Oxygen soon alleviated his dyspnea and he became more manageable. He was slightly febrile with a blood pressure of 110/50 mm. Hg, and a pulse of 140 per minute. His abdomen was moderately distended; the liver was felt 7 cm. below the right costal margin. Tarry stool was present on rectal examination. His hemogram included a hematocrit of 27 per cent and hemoglobin of 8.9 gm. per cent. His blood type was carefully checked and found to be B negative. The non-protein nitrogen was 46 mg. per cent. The Sengstaken tube was removed one day after admission as bleeding had subsided, and transfusions were continued. On the second day jaundice was noted and tracheostomy was done for a purulent bronchitis, since the patient was unable to cough adequately. He was still confused, but had become more cooperative and was cautiously given oral fluids. On the evening of the second day hemorrhage recurred. The patient was taken to the operating room that night and left thoracotomy was performed. The distal esophagus was opened and bleeding varices were suture-ligated. During this procedure 3,500 ml. of blood was administered. Postoperatively the patient became anuric and his blood pressure was maintained only by the use of intravenous norepinephrine. He died thirty-six hours after operation. No autopsy was permitted.

In this young man the source of bleeding could have been a difficult problem to evaluate, but the proper diagnosis was made early. However, incorrect blood typing apparently led to his receiving incompatible blood before his transfer from another hospital. Although his condition was critical, suture of the varices might have proved life-saving, except for the development of renal failure which failed to respond to vigorous therapy with the aid of medical consultants.

Case III. E. F. W. (SLH 77280), a sixty-five year old white retired building contractor, had had two large hematemeses in rapid succession at home. His doctor placed him in a nearby hospital where he received 5,000 ml. of blood in the next eight days. However, as his stools continued to be tarry, he was transferred to St. Luke's Hospital on December 17, 1952. There was no history of alcoholism. The patient had never been jaundiced, although he had noted increase in the size of his abdomen during the past two years. His liver edge was felt 8 cm. below the right rib margin, but no venous distention or capillary hemangiomas were found. The heart was enlarged and a harsh systolic murmur was present. On admission the patient had a hemoglobin of 8.9 gm. per cent, a prothrombin clotting activity of 88 per cent, normal platelet count, and normal bleeding and coagulation times. The albumin-globulin ratio was markedly reversed. Bromsulphalein retention at forty-five minutes was 11.3 per cent, and other liver function tests were moderately abnormal, as was the heparin-protamine titration test. The patient continued to pass tarry stools while receiving vitamin K and other necessary medications. Blood transfusions improved his hemoglobin level to 13.3 gm. per cent. On December 24, 1952, spleno-

renal shunt was performed by the thoracoabdominal approach. The liver was grossly cirrhotic and this was confirmed histologically. Splenic venous pressure was 285 mm. of saline. Postoperatively the patient made a satisfactory recovery. He had no further bleeding until he was readmitted to the hospital on January 19, 1955, for melena of two days' duration. He was found to be in congestive heart failure with auricular fibrillation. His treatment was chiefly medical, being directed toward his cardiac disease. There was no further evidence of melena five days after admission. The patient left the hospital improved, but on June 29, 1956, he died of a myocardial infarction.

This patient initially presented the serious problem of prolonged bleeding and advanced cirrhosis. Although he was a poor surgical risk, successful splenorenal shunt was performed. For two years he remained free of further bleeding. The melena of several days' duration in early 1955 may have been due to recurrent portal hypertension, although the major problem then appeared to be cardiac decompensation. His life was probably prolonged by the shunt operation and good medical management.

COMMENTS

Numerous methods have been advocated in the treatment of esophageal varices, and their relative merits have been evaluated by Lyons and Patten [3]. Transesophagoscopic injection of varices with sclerosing agents has had few adherents. Suture ligation of these veins as a definitive form of therapy was proposed by Crile [4]. Cooley and DeBakey [5] have recommended subtotal esophagectomy, particularly when portal hypertension was due to extrahepatic obstruction. Esophagectomy or esophagogastrectomy may be a final resort when bleeding recurs after a shunt or other procedures have failed. Ligation of the hepatic artery or several branches of the celiac axis has been practiced by some but is not widely accepted.

The most favored type of definitive therapy is the portacaval shunt, revived in 1945 by Whipple [6] and Blakemore and Lord [7]. Linton and Ellis have outlined their approach to the problem as follows [8]. Gastroesophageal balloon tamponade is used to control acute bleeding, but the tube is not used for more than twenty-four hours. Use of the tube also assists in confirming the location of bleeding. Barium swallow may be helpful if the diagnosis is in doubt. Unless there is impending liver failure, transesophageal suture ligation of varices in the distal esophagus and cardia is soon performed. The patient may then be studied and his liver status improved as much as possible before a portacaval shunt is performed.

With various modifications, either direct portacaval anastomosis or splenorenal shunt is usually employed. While the former has more theoretical advantages, the latter appears to have been more widely used, especially if an enlarged spleen was present. Indications for the choice of shunt operations in portal hypertension have been well discussed by Julian [9]. The postoperative mortality rates after shunt operations vary from 6.8 to 25.6 per cent, when both good and poor risk patients are included [10]. In the present series, there were two postoperative deaths after ten shunt operations. This compares closely with three deaths after ten emergency portacaval shunts reported by O'Sullivan and Payne [11]. Although the over-all number of deaths among medically and surgically treated patients presented herein is similar, we are inclined to believe that definitive surgery designed to reduce portal hypertension offers these patients a better chance for longer survival in view of the grave prognosis involved.

SUMMARY

Acute bleeding from esophageal varices is a serious problem with an often fatal outcome. At St. Luke's Hospital, Chicago, there were forty-two admissions of thirty-three patients with acute esophageal bleeding during the years 1950 through 1955. No patients admitted for medical treatment or elective surgery because of remote bleeding were included. Presenting symptoms were hematemesis or melena, or both. Of twenty-five patients with known liver disease, twenty-three had previously bled. Physical findings included, by frequency, hepatomegaly, splenomegaly, jaundice and ascites. Portal cirrhosis was the most frequently found underlying liver disease. The history revealed alcoholism in twelve of the thirty-three cases. A Sengstaken tube was used on seven patients. Following acute bleeding, fourteen patients underwent sixteen surgical procedures, ranging from exploration to major resective and shunt procedures. There were five postoperative deaths and six deaths without surgical intervention. Thus, the mortality rate after acute bleeding in the over-all group was 33.3 per cent. There were two postopera-

tive deaths among ten patients receiving portacaval shunts. While surgical procedures which reduce portal hypertension appear to prolong life in some patients, the prognosis generally is not good after hemorrhage from esophageal varices.

REFERENCES

1. Welch, C. E., Allen, A. W. and Donaldson, G. A. Surgical management of massive acute upper gastrointestinal hemorrhage. *New England J. Med.*, 252: 921, 1955.
2. Nachlas, M. M., O'Neil, J. E. and Campbell, A. J. A. Life history of patients with cirrhosis of the liver and bleeding esophageal varices. *Ann. Surg.*, 141: 10, 1955.
3. Lyons, C. and Patten, T. B. Bleeding esophageal varices. *Surgery*, 39: 540, 1956.
4. Crile, G. Transesophageal ligation of bleeding esophageal varices. *Arch. Surg.*, 61: 654, 1950.
5. Cooley, D. A. and DeBakey, M. E. Subtotal esophagectomy for bleeding esophageal varices. *Arch. Surg.*, 68: 854, 1954.
6. Whipple, A. O. Problem of portal hypertension in relation to hepato-splenopathies. *Ann. Surg.*, 122: 449, 1945.
7. Blakemore, A. H. and Lord, J. W., Jr. Technic of using Vitallium tubes in establishing portacaval shunts for portal hypertension. *Ann. Surg.*, 122: 476, 1945.
8. Linton, R. R. and Ellis, D. S. Emergency and definitive treatment of bleeding esophageal varices. *J. A. M. A.*, 160: 1017, 1956.
9. Julian, O. C. Choice of operation in portal hypertension with varices. *Surg., Gynec. & Obst.*, 100: 753, 1955.
10. Hamilton, J. E. The management of bleeding esophageal varices associated with cirrhosis of the liver. *Ann. Surg.*, 141: 637, 1955.
11. O'Sullivan, W. D. and Payne, M. A. The emergency portacaval shunt. *Surg., Gynec. & Obst.*, 102: 668, 1956.

"*Liver Abscess.*—As already explained, such a complication usually results from an infective embolus or thrombus which has been carried along the portal vein. It may possibly, however, be caused by infection carried along the lymphatic vessels. Though these do not connect directly with the liver, the communication may be made through the lymphatics of the abdominal wall; or, what is perhaps more common, the suppurative process may extend directly from an abscess. Whichever be the origin, such abscesses are most often multiple. The symptoms are those already described as arising when the portal veins become infected. In addition, there may be more or less jaundice. There is apt to be delirium, and the obstinate dryness of the tongue is especially noticeable. The treatment is operation, but the results are apt to be discouraging. . ." (From: Bryant, J. D. and Buck, A. H. American Practice of Surgery. Baltimore, 1910. William Wood & Co., division of Williams & Wilkins Co.)

Delayed Postgastrectomy Hemorrhage from Ruptured Traumatic Aneurysm

CLIFTON F. WEST, JR., M.D., *Philadelphia, Pennsylvania*

From the Surgical Service of Dr. Gilson Colby Engel, Lankenau Hospital, Philadelphia, Pennsylvania.

WITH the proficiency of subtotal gastrectomy for complicated duodenal ulcers steadily increasing, mortality rates have decreased and now average less than 2 per cent in most clinics. However, the problem of the extensive eroding duodenal ulcer of long-standing which has not only scarred but also shortened the duodenum still remains a problem. The following case of delayed postgastrectomy hemorrhage emphasizes another of the pitfalls which one may encounter in dealing with just such an ulcer.

CASE REPORT

H. L., a fifty-four year old man, was admitted to the Lankenau Hospital on December 27, 1950, with a chief complaint of nausea and vomiting associated with epigastric distress. The past medical history showed that the patient had vague indigestion and epigastric pain relieved by food and antacids for approximately four years. Physical examination revealed a moderately dehydrated fifty-four year old male with moderate deep tenderness in the right upper quadrant of the abdomen. Laboratory studies showed no abnormalities. Nasogastric suction was instituted and intravenous fluids were given. On the fifth day after admission an upper gastrointestinal series showed a large ulcer crater of the first portion of the duodenum. The patient was placed on a strict medical regimen and discharged fourteen days after this first admission.

The patient did fairly well at home for four months until he was readmitted to the Lankenau Hospital with recurrent epigastric pain radiating through to the back. Examination at this time revealed some epigastric fullness and tenderness in the right upper quadrant of the abdomen. Laboratory examinations including a prothrombin time determination were all normal. A repeat upper gastrointestinal series showed a moderate amount of obstruction at the lower end of the stomach with a constantly deformed and distorted duodenum. Exploration was performed and a large obstructing duodenal ulcer was found. There was marked edema around the head of the pancreas and the first portion of the duodenum. The rest of the abdominal exploration was essentially normal. Subtotal gastrectomy was performed and approximately the distal two-thirds of the stomach was removed. An antecolic Polya II type resection was carried out. A note in the operative report described the edema around the duodenum and stated that the duodenum was sectioned just at the level below the ulcer. The duodenal stump was closed with the usual two-layer closure and was buttressed into the head of the scarred pancreas. The pathologic report described a large benign duodenal ulcer, approximately 1.5 cc. in diameter, which had apparently perforated through into the pancreatic capsule. The patient did well postoperatively and was tolerating a soft gastric diet by the fifth postoperative day. He was discharged from the hospital ten days after admission on a light gastric diet, with no complaints.

The patient continued to do well at home until his third hospital admission in May, approximately five weeks following operation. He was readmitted this time with massive hematemesis. He had noted tarry bowel movements two days prior to admission. At this time the patient was obviously in shock with a blood pressure of 70/40 mm. Hg and a pulse of 120. Abdominal findings showed a well healed scar in the right upper quadrant of the abdomen with no masses or tenderness. Rectal examination showed the rectum to be filled with old blood. Hemoglobin was 7 gm. per cent with 2 million red blood cells. The patient was given 1,500 cc. of blood through a cannulated vein in his ankle and taken to the operating room eight hours after admission to the hospital. The abdomen was reopened through the previous incision. The peritoneal cavity was found free of any fluid or exudate. The duodenal stump, which had been buried into the head of the pancreas, was covered with a moderate amount of fibrous tissue but otherwise appeared normal. The gastric stump was dis-

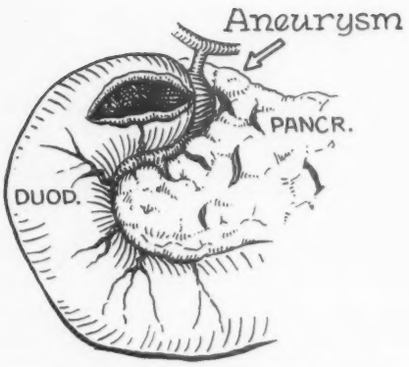


FIG. 1. Diagrammatic drawing of aneurysm.

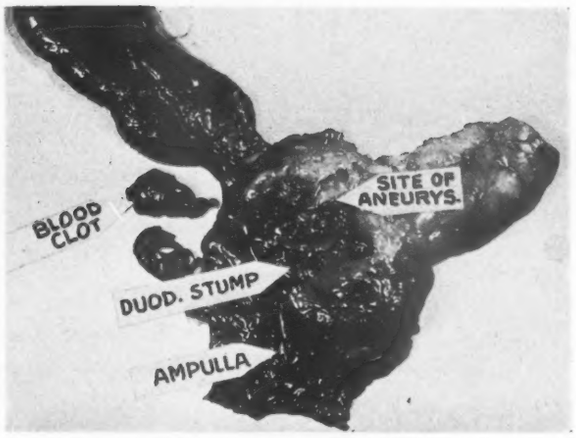
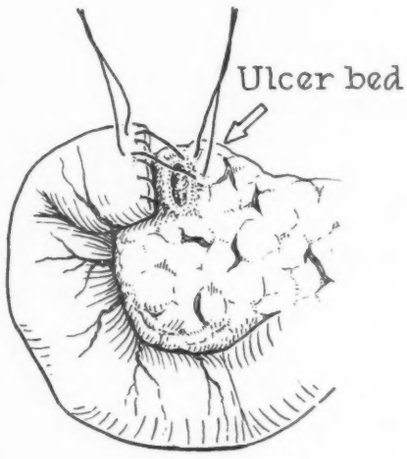


FIG. 2. Autopsy specimen.

tended and obviously filled with blood. A longitudinal gastrostomy incision was made just above the recently performed gastrojejunal anastomosis. There appeared to be no area of frank hemorrhage in the stomach itself, and there were no active bleeding sites at the anastomosis. However, there were thought to be several suspicious areas around the anastomotic ring, and a continuous running chromic catgut suture was used to control any possible bleeding from this area. No evidence of esophageal varices was noted. The bleeding seemed to have ceased and the gastrostomy site was closed. The patient was returned to his room with a blood pressure of 130/70 mm. Hg and a pulse of 110. He did fairly well for the next twelve hours except for some difficulty in keeping the Levin tube open due to old blood clots from the gastrointestinal tract. However, approximately forty-eight hours after surgery, the patient again started vomiting bright red blood. At this time the Levin tube was removed and an esophageal tamponade tube was passed with the feeling that the patient might possibly be bleeding from a ruptured esophageal varix. The patient was given repeated transfusions and over the next forty-eight hours seemed to hold his own. However, four days later he had another massive episode of hematemesis during which he vomited the tube, and in spite of numerous transfusions he died. The patient had actually received a total of 30 pints of whole blood during this last admission, the last eight of which were fresh blood.

The autopsy findings pertinent to this case showed the peritoneal cavity to be free of any blood or fluid. The entire small and large bowel and gastric remnant were acutely distended with fresh and old blood. The esophagus was found to contain no varices. The gastric stump showed the recent gastrostomy site with no evidence of any recent bleeding point. The gastric remnant, the entire duodenum and pancreas were removed as an *en bloc* specimen. When the duodenum was opened, at first there appeared to be chronic and sloughing invaginated duodenal tissue. However, on closer examination this was found to be an organizing blood clot lying embedded in the duodenal stump



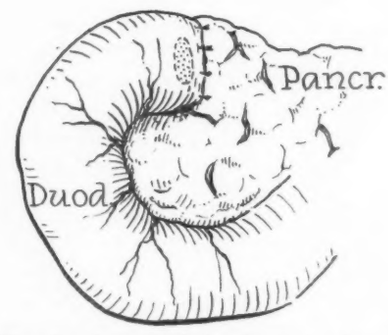


FIG. 3. Duodenal stump being buttressed against ulcer bed.

2 cm. proximal to the ampulla of Vater. Upon removing the blood clot an opening was found, approximately 3 mm. in diameter, which extended from the edge of the duodenal stump into the gastroduodenal artery. The specimen was dissected further and a berry-sized traumatic aneurysm was seen arising from the gastroduodenal artery. This had apparently ruptured into the duodenum causing the massive hematemesis. (Figs. 1 and 2.) It was believed at postmortem examination

that the aneurysm was the result of one of the sutures which had nicked the side of this main artery while an attempt was being made to bury the stump into the head of the pancreas.

COMMENTS

Much has been written in the literature concerning the management of the duodenal stump. Most authors, and rightly so, emphasize the seriousness of postoperative leakage and breakdown of the suture line with its so-called blown stump. This still accounts for the major mortality associated with the difficult ulcer case. The dangers of encroaching upon the common duct and the pancreatic ducts have likewise been pointed out.

Hemorrhage itself following subtotal gastrectomy usually occurs in the immediate twenty-four–hour postoperative period and is due to bleeding from the gastroenterostomy site. However, it may be delayed eight to ten days postoperatively and is usually caused by sloughing of the strangulated mucous membrane at the suture line. If this is persistent or becomes massive, immediate gastrostomy with either a continuous over and over or locking catgut suture around the anastomosis is all that is required. Delayed hemorrhage from an ulcer left *in situ* in the duodenum following adequate subtotal gastrectomy has been reported but must be exceedingly rare. This unusual case of delayed hemorrhage caused by ruptured traumatic aneurysm of the gastroduodenal artery into the buried duodenal stump has not been recorded as far as could be determined. If one reviews the arterial blood supply of the duodenum and the head of the pancreas, it is found that the anterior and posterior branches of the superior pancreaticoduodenal artery freely anastomoses with the inferior pancreaticoduodenal artery and lies just within the pancreatic capsule. This may actually be demonstrated in patients who have a normal-appearing pancreatic capsule with no edema of the head of the pancreas. However, when there is an eroding ulcer with marked scarring and edema, the anatomy of the vessels in this area is quite obscure. When one is concerned with closure of the duodenal suture line, he frequently, as has been advocated by many authors, attempts to bury the stump into the head of the pancreas; or, if a chronic ulcer bed is present, he is tempted to use the edge of the ulcer bed to buttress the suture line. (Fig. 3.) This is what was performed in the case reported herein. If one inadvertently happens to pierce a branch of one of the major arteries in this area, he is almost invariably forced to tie down the suture to obtain hemostasis. However, the wisdom of using the same suture to roll the duodenal suture line into the head of the pancreas must be questioned in view of this case.

Traumatic aneurysm produced in this case by injury to the major gastroduodenal artery was completely obscured at the second operation due to the fact that the duodenal stump had been rolled over anterior to the wall of the vessel. The site of the hemorrhage was obscured even at the time of postmortem examination until the duodenal stump was opened and the clot lifted away from the lumen of the stump and away from the site of the ruptured aneurysm. However, if one keeps this possibility in mind in any case of delayed hemorrhage following subtotal resection for difficult duodenal ulcer or in a case in which the stump has been buttressed against the pancreas, this unfortunate outcome might be prevented.

SUMMARY

An unusual case of delayed postgastrectomy hemorrhage is reported.

Extreme care must be taken in reenforcing the duodenal suture line to the pancreatic capsule or the adherent ulcer bed. The wisdom of excluding the adherent or penetrating duodenal ulcer should be stressed.

Finally, if faced with persistent hemorrhage in the postoperative period following gastrectomy and after exploring the anastomotic site no obvious bleeding area is found, one should always consider the possibility of the duodenal stump as the source of hemorrhage.

REFERENCES

1. Kleitsch, W. P. Anatomy of the pancreas. *Arch. Surg.*, 71: 795–802, 1955.
2. Mayfield, R. C. and Abramson, P. D. Use of catheter duodenostomy in subtotal gastrectomy. *Am. J. Surg.*, 90: 998–1001, 1955.
3. Welch, C. E. and Rodkey, G. V. Method of management of duodenal stump after gastrectomy. *Surg., Gynec. & Obst.*, 98: 376–379, 1954.
4. McKittrick, L. S., Moore, F. D. and Warren, R. Complications and mortality in subtotal gastrectomy for duodenal ulcer. *Ann. Surg.*, 120: 531–561, 1944.
5. McNealy, R. W. Technical details in management of duodenum in gastric resection. *S. Clin. North America*, 26: 200–209, 1946.
6. Ross, F. P. and Warren, R. Safeguards in gastric resection for duodenal ulcer, *New England J. Med.*, 245: 475–481, 1951.

Surgical Importance of Bile Duct Adenoma of Liver

HUGH F. LUDDECKE, M.D. AND ILL LYONG YOON, M.D., *Morristown, New Jersey*

From the Department of Pathology, Morristown Memorial Hospital, Morristown, New Jersey.

BENIGN neoplasms of the liver are rare [1,2,15] and usually silent. They are found incidentally at autopsy or at laparotomy performed for other reasons. It is not our purpose to define their histogenesis or pathogenesis but rather to emphasize their surgical importance. Benz, Baggenstoss [3] and Popper and Schaffner [10] have pointed out previously, and we are in agreement, that the gross appearance of these lesions can simulate metastatic carcinoma and it is for this reason that we are presenting herein the cases of two patients in whom bile duct adenomas were found.

FIG. 1. Apparently circumscribed oval tumor in liver (Case 1).

Benign neoplasms of the liver of a non-mesenchymal type can be classified as bile duct type (cholangiocellular), hepatic cell type (hepatocellular) and mixed type. Many authors consider that all neoplasms of this group are really hamartomas [7,10,11]. Also to be classified among the benign tumors of the liver are hemangiomas but as these do not have the gross appearance similar to or suggestive of metastatic carcinoma they will not be further considered herein. Adrenal rest tumors and "focal cirrhosis" [3] are probably additional lesions which should be included among those which can simulate metastatic carcinoma but we have had no experience with these.

We have had an opportunity recently to study two cases of bile duct adenoma found incidentally at autopsy, one in a patient who died of bronchogenic carcinoma and the other in a patient who died of acute myocardial infarction.

CASE REPORTS

CASE 1. A fifty-eight year old white man was admitted because of severe dyspnea. With this he had had backache with pain radiating into his lower extremities for four days. There had been a 50 pound loss of weight in the past three months. The patient died sixteen hours after admission.

At autopsy, a pale yellow-white oval, partially circumscribed tumor 10 by 8 by 6 cm. having origin in the right upper main stem bronchus was found. The peribronchial and mediastinal nodes were enlarged up to 5.0 cm. in diameter and flat, round metastatic nodules were found in the serosa of jejunum, 3.0 cm. and 5.0 cm. in diameter. Cylindric antemortem thrombi were demonstrated in the pulmonary veins draining the right upper lobe and in the common iliac arteries.

The liver weighed 2,560 gm. The outer surface was smooth, glistening, and pale brown; an apparently circumscribed spherical tumor was found in the inferior aspect of the right lobe 1.0 cm. in diameter. (Fig. 1.) This was visible through the capsule and we considered it to be metastatic carcinoma from its gross appearance.

The microscopic sections showed the tumor of the bronchus to be an epidermoid carcinoma. There were metastases in the jejunum, mediastinal, periaortic and peripancreatic lymph nodes. The thrombi found in the pulmonary veins and iliac arteries showed many clusters of tumor cells similar to those seen in the bronchogenic carcinoma.

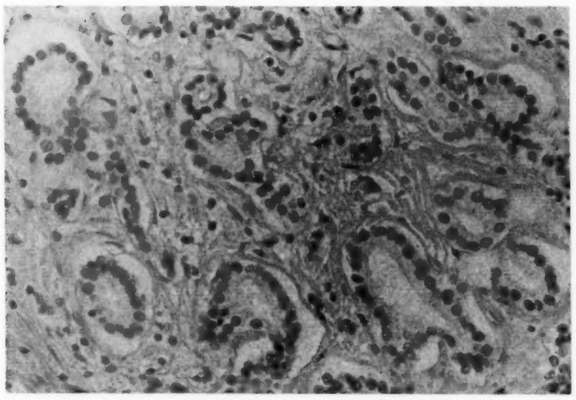


FIG. 2. Sections of the liver nodule show innumerable duct structures of a biliary type imbedded in a dense fibrous matrix (Case 1). Hematoxylin and eosin stain. Original magnification, × 430.

The sections of the liver nodule revealed innumerable, uniform duct structures of a biliary type imbedded in a dense fibrous matrix. These were lined by a single row of columnar cells with basally placed, round, uniformly stained nuclei. (Fig. 2.) None of these lining cells revealed pleomorphism or mitotic activity and the duct spaces occasionally contained a minute amount of acellular, granular eosinophilic material. In the fibroconnective tissue were only rare lymphocytes. The adjacent hepatic tissue and other random sections of the liver showed no evidence of cirrhosis.

Pathologic diagnoses were as follows: epidermoid carcinoma in right upper mainstem bronchus with metastases to jejunum, mediastinal, periaortic, peripancreatic lymph nodes; tumor thrombi in pulmonary veins and (by embolization) iliac arteries; and bile duct adenoma of liver.

CASE II. A sixty-eight year old white man was admitted with severe dyspnea. An hour prior to admission pain suddenly developed in the left anterior part of the chest radiating to the left arm. The patient had been in another hospital recently where he was said to have had an aortic valvular lesion. Since then he had been taking digitalis. On admission he was gasping, cyanotic and his chest contained many coarse moist rales. The heart sounds were inaudible and blood pressure was unobtainable. Fifteen minutes after admission he died.

At autopsy a fresh myocardial infarct in the posterior wall of the left ventricle was found. The liver weighed 1,650 gm. The capsule was smooth but there were two apparently well circumscribed non-elevated gray-white tumors seen through the capsule, one 0.3 cm. and the other 0.7 cm. in diameter. (Fig. 3.) The cut surfaces of these tumors were gray, firm and dry. The tumors were entirely separate from each other.

FIG. 3. Well circumscribed non-elevated gray-white tumor in liver (Case II).

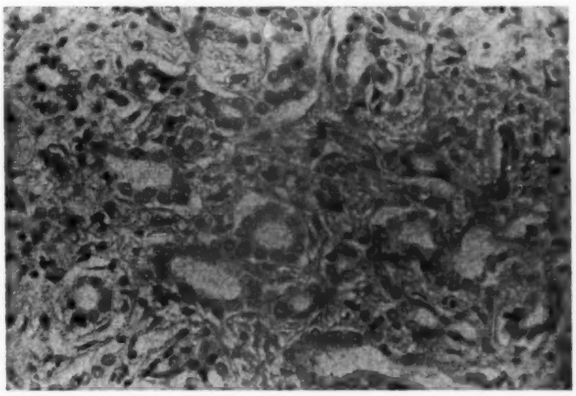


FIG. 4. Liver nodule is composed of rather dense fibroconnective tissue in which were numerous small, slightly irregular duct structures (Case II). Hematoxylin and eosin stain. Original magnification, × 430.

The microscopic sections of the liver showed the nodules to be composed of rather dense fibroconnective tissue in which were numerous small, slightly irregular duct structures lined with uniform columnar or cuboidal cells. (Fig. 4.) The nuclei were oval or round and uniform in size. No pleomorphism or mitotic activity was seen. The hepatic tissue showed no evidence of cirrhosis. The sections of the heart confirmed the gross diagnosis of acute myocardial infarction.

Pathologic diagnoses were myocardial infarction, recent, posterior; and bile duct adenomas (two) of the liver.

COMMENTS

At the time of autopsy the gross impression of a single nodule in the liver was that of a metastatic deposit from a bronchogenic carcinoma (Case 1). A photograph taken from our file of a gross specimen of a metastatic carcinoma in the liver from another case of epidermoid carcinoma of the bronchus is

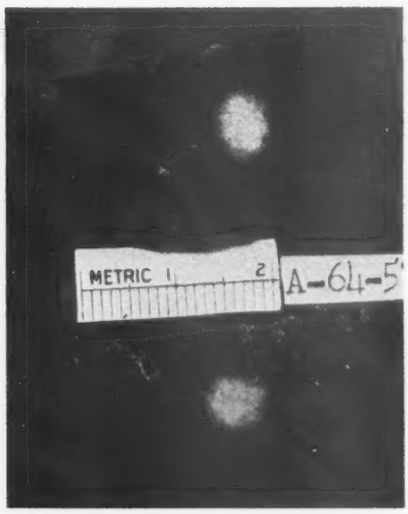


FIG. 5. A photograph taken at random from our file showing metastatic carcinoma in the liver from bronchogenic carcinoma. Note the similarity to Figures 1 and 3.

reproduced (Fig. 5) for purposes of comparison. The microscopic sections revealed an entirely benign bile duct adenoma. In the second patient (Case II) there were two bile duct adenomas thus making it even more likely to be grossly confused with metastatic disease. If one were to encounter this type of nodule at laparotomy for cancer anywhere in the gastrointestinal tract it could easily be mistaken for metastatic tumor and hence result in the decision that the case was inoperable.

Although benign neoplasms of the liver are infrequent, usually small [*12*], and asymptomatic, their chief importance is that they might be grossly mistaken for metastatic carcinoma. It has been reported that these tumors can occasionally grow to a large size and produce symptoms by pressure. In the literature there are such cases of patients who have been successfully treated with surgery [*5,8,9*]. Another consideration is whether these neoplasms can become malignant. It has been stated [*6*] that these bile duct adenomas are potentially malignant and that all primary hepatic neoplasms, benign and malignant and discussed under many names, are merely different phases or stages of one pathologic process [*6*]. From our study of these two cases and from the primary hepatic carcinomas which we have studied we believe it would be difficult to establish such a relationship. Our patients certainly presented no cytologic evidence of malignancy. There were circumscribed lesions but no capsules could be defined. Warvi [*16*] and Boyd [*4*] require encapsulation of a benign process of this type as a criterion of adenoma. Others [*14*] including ourselves, do not believe this is necessary. These tumors apparently grow by expansion with resultant compression of surrounding liver substance; this obtained in our cases and has been referred to by Anderson [*1*]. Generally malignant primary neoplasms of the liver grow by replacing or invading adjacent hepatic tissue.

Non-neoplastic hyperplastic nodules composed of fibrous tissue (scar) and biliary ducts (reparative) are more difficult to separate exactly from adenomas. However the reparative nodules would be more apt to be multiple and associated with some other recognizable liver disease such as cirrhosis, focal or otherwise, parasitic infestation, chemical poisoning, sepsis [*13*] or other diseases which could conceivably cause focal necrosis.

SUMMARY

Two cases of bile duct adenoma found at autopsy are reported. The gross similarity of this lesion to metastatic carcinoma is stressed and it is urged that before deciding on the significance of a liver nodule at surgery it be examined by a quick frozen section.

REFERENCES

1. ANDERSON, W. A. D. Pathology, p. 884. St. Louis, 1948. C. V. Mosby Co.
2. ACKERMAN, L. V. Surgical Pathology, pp. 384–386. St. Louis, 1953. C. V. Mosby Co.
3. BENZ, E. J. and BAGGENSTOSS, A. H. Focal cirrhosis of liver. Its relation to the so-called hamartoma (adenoma, benign hepatoma). *Cancer*, 6: 743–755, 1953.
4. BOYD, W. Pathology for the Surgeon, 17th ed., p. 263. Philadelphia, 1955. W. B. Saunders Co.
5. DELANNOY, E. and LAGACHE, G. Adenomes biliaires kystiques. À propos de 5 observations personnelles. *Arch. d. mal. de. l'app digestif.*, 36: 452–456, 1947.
6. FRANKLIN, R. G. and DOWNING, C. F. Primary liver tumors. *Am. J. Surg.*, 73: 390–395, 1947.
7. KAY, S. and TALBERT, P. C. Adenoma of the liver, mixed type (hamartoma). Report of two cases. *Cancer*, 3: 307–315, 1950.
8. LONGMIRE, W. P. and SCOTT, H. W., JR. Benign adenoma of the liver (successful surgical resection of tumor involving both right and left lobe). *Surgery*, 24: 983–988, 1948.
9. PACKARD, G. B. and STEVENSON, A. W. Hepatoma in infancy and childhood, discussion and report

of patient treated by operation. *Surgery*, 15: 292–306, 1944.
10. POPPER, H. and SCHAFFNER, F. Liver: Structure and Function, pp. 587–592. New York, 1957. McGraw Hill-Book Co.
11. ROSSO, D. C. Una particolare alterazione epatica, la fibroadenomatosi biliare. *Arch. Ital. di Anat. E. Istolog. Patol.*, 28: 189–194, 1954.
12. STOUT, A. P. Human Cancer, p. 142. Philadelphia, 1932. Lea & Febiger.
13. SCHRAGER, V. L. Surgical aspects of adenoma of the liver. *Ann. Surg.*, 105: 33–43, 1937.
14. STUMPF, H. H. and LIBER, A. F. Hepatocellular adenomatosis: report of a case with liver function studies. *Am. J. Med.*, 17: 887–890, 1954.
15. WARVI, W. N. Primary neoplasms of the liver. *Arch. Path.*, 37: 367–382, 1944.
16. WARVI, W. N. Primary tumors of liver. *Surg., Gynec. & Obst.*, 80: 643–650, 1945.

"De Mondeville was one of four body-surgeons of Philip the Fair, and he gave lectures on anatomy at the University of Montpellier. He was unorthodox again in disregarding the accepted practice of reducing to a starvation diet patients who had been injured. He advised that they should be assisted to recovery by good food and good wines. In addition, the 'music of viols and a ten-stringed psaltery' would help in maintaining the patient's morale. De Mondeville was insistent on the importance of keeping the patient's spirits up, and the better to stress this point he wrote cheerfully that he was quite prepared to forge letters announcing the death of the patient's enemies or the exaltation of an ailing cleric to a bishopric, if he thought it would promote recovery." (From: GRAHAM, H. Surgeons All, p. 103. New York, 1957. Philosophical Library, Inc.)

Hendrik van Roonhuyze (1625?) performed a casarean section several times, or so we are told. His "Heelkonstige Aanmerkkinger" (1663) has been described as the first work on operative gynecology in the modern sense. It is illustrated with unique copper plates, showing his mode of incision in cesarean section, and contains case reports of ectopic pregnancy and rupture of the uterus. (T. S. W.)

Volvulus of the Transverse Colon

SCOTT J. BOLEY, M.D., *Brooklyn, New York*

From the Department of Surgery, Jewish Hospital of Brooklyn, Brooklyn, New York.

VOLVULUS of the transverse colon alone is rare, only twenty-four such cases being previously recorded. Kallio [6], reviewing the literature prior to 1932, reported eighteen cases including two of his own. Figiel and Figiel [3] in 1954 added three more from the literature, one of their own, and one from a personal communication. Zaslow and Orloff [9] reported a true volvulus of the transverse colon as a postoperative complication. Other authors have reported cases as transverse colon volvulus but these have involved portions of ascending or descending colon.

Volvulus of the colon accounts for 3 per cent of large bowel obstructions in the United States (although in Scandinavian and Eastern European countries this figure may rise to 30 per cent). Since torsions of the transverse colon constitute only a small fraction of 1 per cent of these, a rough estimation of their incidence would be one in every 30,000 cases of large bowel obstruction.

Gerwig [5] has listed the following factors, any or several of which may be concerned in the etiology of colon volvulus: (1) elongation of the mesentery with a freely movable bowel, (2) absence of the mesentery with a mobile bowel, (3) closely approximated points of fixation, and (4) adhesions from previous surgery. The short mesentery of the transverse colon and the well separated points of fixation at the hepatic and splenic flexures mitigate the occurrence of torsion in the transverse colon alone. Thus the rarity of this lesion is not difficult to comprehend.

Certain predisposing factors apparently have been present in most of the cases previously reported and are also found in our own. These are: (1) some abnormal point of fixation, i.e., adhesions, or (2) colonic obstruction distal to the volvulus. Adhesions, either secondary to hernias or abdominal operations, were described in over 25 per cent of the cases reported.

Finsterer [4] in 1931 first mentioned the association of obstructing lesions of the left colon and volvulus of the transverse colon. One of his patients had a carcinoma of the sigmoid as well as a volvulus. Martin and Ward [7], Roos and Curschman [6], and Becker [1] all reported distal obstructions including sigmoid volvulus and inflammatory stricture of the colon. Wilson, Desforges, Dunphy, and Campbell [2,8] reported on eight patients with cecal volvulus, four of whom had left colon lesions, but did not venture to explain the association. The distention and lengthening of the colon proximal to an obstruction might account for the development of torsion whereas under normal circumstances this could not occur. Certainly some factor must be present to overcome the natural protection against a volvulus of the transverse colon and produce this most uncommon condition.

The radiological diagnosis of this lesion can be made by barium enema, as demonstrated in the two cases reported. The value of an exact preoperative diagnosis in the operative approach to any large bowel obstruction needs no comment, and in transverse colon volvulus the knowledge obtained well warrants the time needed for roentgenographic studies.

The following cases are presented in order to supplement the limited literature on this subject and to emphasize that some causative agent is almost always present. It is essential that this etiologic factor be found in order to assure adequate surgical therapy.

CASE REPORTS

CASE 1. R. L., an eighty-two year old white woman, was admitted to the hospital with a three day history of obstipation, increasing abdominal distention, crampy abdominal pain, anorexia and vomiting. Prior to this episode the patient had been

free of any abdominal or gastrointestinal complaints. Thirty years prior to admission she had had a left inguinal herniorrhaphy at which time a right inguinal hernia had been noted. The latter had increased in size but was always reducible. There was no other history of previous surgery or intra-abdominal disease.

Physical examination revealed an emaciated, mildly dehydrated, elderly woman in no acute distress. The chest findings were normal except for signs of arteriosclerotic heart disease commensurate with the patient's age. The abdomen was markedly distended, minimally tender diffusely, and a loop of bowel was easily outlined in the right lower quadrant. No masses or muscle guarding was noted and the bowel sounds were markedly hypoactive. Pelvis and rectum revealed no abnormalities, the latter being empty. A large reducible right inguinal hernia was found.

Laboratory studies were normal except for a blood urea nitrogen of 58 mg. per 100 cc. A plain film of the abdomen was interpreted as showing a markedly distended cecum with moderate distention of the ascending colon. The diagnosis at this time was an obstructing lesion of the colon, most probably in the area of the hepatic flexure.

Several hours after admission the patient had two spontaneous bowel movements. In view of this and the patient's generally fair condition, medical decompression with enemas and Cantor tube was initiated. Following this the patient had several large watery bowel movements over a period of forty-eight hours with clinicial evidence of almost complete relief of distention. Serial X-ray studies corroborated the effective decompression of the distended large bowel.

Three days after admission the patient again presented clinical evidence of intestinal distention with a tense, tympanitic abdomen and an easily outlined loop of bowel in the right lower quadrant of the abdomen. An emergency barium enema was interpreted as showing a volvulus of the transverse colon with an adhesion between this portion of the colon and the right inguinal hernial sac. The gas shadow previously interpreted as cecum was shown to be distended transverse colon.

Accordingly laporatomy was performed and at operation the x-ray diagnosis was corroborated. The transverse colon was on a relatively long mesocolon and its mobility was further enhanced by a low fixation of the hepatic flexure. A volvulus with a 270 degree twist was present involving the transverse colon, and its fulcrum was an adhesion between this bowel and the right inguinal hernial sac. (Fig. 1.) The hernia was sliding in nature involving the right fallopian tube and ovary. The volvulus was relieved and all adhesions lysed. No attempt was made to repair the sliding hernia at this time due to the patient's condition.

The postoperative course was uneventful except for pneumonia in the right lower lobe which responded readily to therapy. Passage of flatus occurred three days after surgery and spontaneous bowel movements on the fifth day. The patient was discharged to a nursing home three weeks postoperatively.

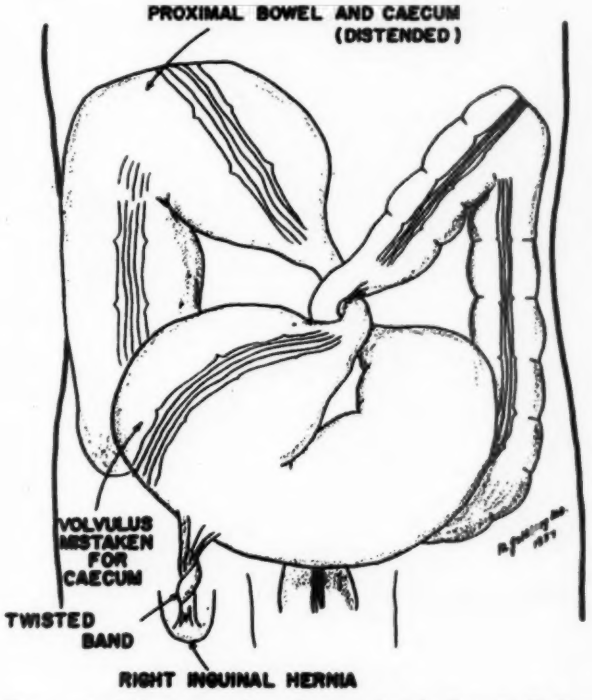


FIG. 1. Artist's conception of operative findings in R.L., Case 1. The adhesion to the right inguinal hernial sac is the fulcrum around which the transverse colon has twisted.

Comment: In this case a point of fixation at the site of the inguinal hernia combined with a low hepatic flexure and an abnormally long mesentery to permit the torsion of the transverse colon. Had surgery been performed when the probable diagnosis was an obstructing lesion at the hepatic flexure a cecostomy would have been the accepted procedure. By enabling the correct diagnosis to be made the barium enema permitted the surgeon to attack the real problem.

CASE II. D. H., a sixty-eight year old white man, was admitted to the hospital with a one day history of severe, crampy pain in the right lower quadrant of the abdomen. The onset was acute and followed a two day period of obstipation. For several months prior to this episode he had had mild crampy abdominal pains, but otherwise was asymptomatic. Past medical history was non-contributory.

Physical examination revealed a critically ill, white man in acute distress. Positive findings were limited to the distended abdomen. Tenderness was present in the right lower quadrant, but there was no muscle guarding, rigidity, or rebound tenderness. No masses were palpable, but the distention was more pronounced in the right lower quadrant. Bowel sounds were hyperactive. Rectal examination revealed no abnormalities.

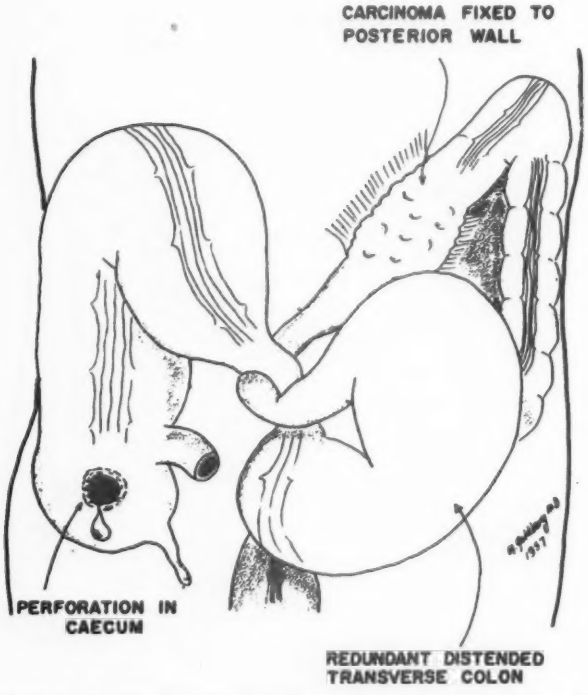


FIG. 2. Operative findings in D. H., Case II. The fixation of the carcinoma posteriorly has approximated the flexures as well as lengthening the transverse colon. This combination created a situation in which volvulus could occur.

Laboratory findings were within normal limits. A plain film of the abdomen revealed marked distention of the cecum, ascending and transverse colon. Small bowel was only minimally distended. The interpretation was a mechanical obstruction of large bowel distal to transverse colon.

A Miller-Abbot tube was passed and attached to Wangensteen suction. An emergency barium enema was interpreted as volvulus of the transverse colon with a possible obstruction in the area of the splenic flexure. After preoperative preparation with intravenous fluids, exploratory celiotomy was performed.

At operation a constricting carcinoma of the transverse colon was found, just proximal to the splenic flexure and adherent to the posterior parietal peritoneum almost in the midline. (Fig. 2.) A volvulus of the transverse colon was present proximal to the carcinoma. The bowel involved in the volvulus was distended to five times normal size and had a 270 degree twist. There was no obvious impairment to the blood supply. The cecum and ascending colon were markedly distended and the former showed early signs of infarction and gangrene. Free amber fluid was present in the peritoneal cavity.

After mobilizing the colon from ascending to descending colon the volvulus was untwisted. The cecum was aspirated and a tube cecostomy performed. The latter was brought out through a stab wound in the right lower quadrant. A Rankin obstructive resection was then performed, removing the left half of the transverse colon and the splenic flexure.

Postoperatively the abdominal distention persisted although the cecostomy began functioning on the first day. On the fifth postoperative day singultus developed and with coughing, evisceration occurred. The wound was resutured in the operating room with through-and-through silk sutures. The same night the patient pulled out the cecostomy tube.

Following this the postoperative course was stormy but on the fifteenth day all sutures were removed. On the twenty-seventh day the patient was discharged with the abdominal wound well healed and the colostomy functioning. Closure of the colostomy was deferred until the patient's general condition could be improved.

Comment: This case represents a volvulus of the transverse colon which occurred proximal to an obstructing neoplasm of the left colon. A secondary factor was the fixation of the tumor near the midline, thereby approximating the two flexures. Again, the barium enema provided the accurate preoperative diagnosis.

SUMMARY

The literature on volvulus of the transverse colon has been reviewed. Two new cases of this rare entity are reported. The author emphasizes that etiologic factors are almost always present and must be found in order to insure adequate surgical therapy. Emergency barium enema is recommended in obstructions of the large bowel.

REFERENCES

1. BECKER, W. F. Acute obstruction of the colon: an analysis of 205 cases. *Surg., Gynec. & Obst.*, 96: 677, 1953.
2. DESFORGES, G. and WILSON, H. Volvulus of the cecum. *Am. J. Surg.*, 86 116, 1953.

3. FIGIEL, L. S. and FIGIEL, S. J. Volvulus of the transverse colon. *Radiology*, 63: 832, 1954.
4. FINSTERER, H. Die Chirurgie des Dickdarms. *Arch. klin. Chir.*, 164: 349, 1931.
5. GERWIG, W. H., JR. Volvulus of the colon. *S. Clin. North America*, 35: 1395, 1955.
6. KALLIO, K. E. Über Volvulus Coli Transversi. *Acta chir Scandinav.*, 70: 39, 1932.
7. MARTIN, J. D., JR. and WARD, C. S. Megacolon associated with volvulus of transverse colon. *Am. J. Surg.*, 64: 412, 1944.
8. WILSON, H. E., DESFORGES, G. and DUNPHY, H. G. Volvulus of the cecum. *Arch. Surg.*, 68: 593, 1954.
9. ZASLOW, J. and ORLOFF, T. Volvulus of the transverse colon occurring as a postoperative complication. *Am. J. Surg.*, 87: 780, 1954.

"At the beginning of the sixteenth century, though medicine was still bigoted, cloistered and bound by tradition, a few signs of new life were beginning to appear. There were a number of schools where an ambitious lad could go to learn his Galen and perhaps some of the teachings of the Arabian physician Avicenna and the Persian Rhazes. A few books on anatomy were beginning to appear, though all followed the ancient teaching and all of them were bad by Renaissance standards. It had been possible for some time to get a degree in medicine as in other subjects, and in the fourteenth century holders of such a degree had begun to be called doctors. There was little if any improvement in surgery, however, for there was no real knowledge of anatomy. Surgery had been born in superstition; it grew in ignorance." (From: YOUNG, A. Scalpel: Men Who Made Surgery, p. 9. New York, 1956. Random House.)

"The treatment of syphilis is twofold: A routine course, covering the first three years or so of the disease, by which we hope to rid the patient of his malady; and beyond this, perhaps simultaneously, perhaps subsequently, the treatment required to conquer the symptoms from which the patient happens to suffer." (From: BRYANT, J. D. and BUCK, A. H. American Practice of Surgery. Baltimore, 1910. William Wood & Co., division of Williams & Wilkins Co.)

Carcinoma of the Common Bile Duct

DONALD C. GEIST, M.D., *Philadelphia, Pennsylvania*

CARCINOMA of the common bile duct occurs rarely. The following report of a patient with this disease illustrates its clinical features and demonstrates rather long survival from a palliative operation.

CASE REPORT

F. G., a seventy-five year old white woman, was admitted to the Misericordia Hospital, Philadelphia, Pennsylvania, on August 6, 1954. She complained of jaundice, nausea, itching and pain in the back. Three months prior to her admission, she began to have nausea and discomfort in the back, which became worse after eating fatty foods. Two weeks before her admission, jaundice was noticed and severe itching of the skin had begun. All of her symptoms became more severe as time passed and, in addition, she noticed that her urine was very dark in color and her bowel movements were light and clay-colored. She was known to have had a single gallstone for fifteen to twenty years. A benign rectal polyp was removed in 1949. There was x-ray evidence of diverticulosis of the colon and chronic calculus cholecystitis in 1953. Nothing else of significance was noted in her past medical, family or social history.

Physical examination revealed an elderly white female with moderately severe jaundice of the skin and sclerae, and numerous scratch marks on her skin. Her systolic blood pressure was 160 mm. Hg, and the diastolic 90 mm. Her temperature was 99, pulse rate 80, and respiratory rate 20. There was moderate tenderness in the epigastrium and the right upper quadrant of the abdomen. The liver was palpable below the costal margin and tender to palpation. There was partial ankylosis of the right knee from a previous arthritis. No other physical abnormalities were found.

All laboratory examinations were normal with the following exceptions: The serum bilirubin varied between 5.6 and 11.3 mg. per cent. The icteric index was 42.6, cephalin flocculation test 4 plus, and thymol turbidity 7 units. Her urine was positive for bile and negative for urobilinogen. The feces were positive for urobilin. An upper gastrointestinal x-ray study was normal.

The patient was operated upon on August 25, 1954. The gall bladder was markedly dilated with white bile and contained a stone the size of a hen's egg. The fundus of the gall bladder was markedly adherent to the hepatic flexure of the colon and to the duodenum. It was considerably thickened and fibrosed and there was a constriction near the middle of the fundus. In the region of the junction of the cystic and common hepatic ducts, there was a hard, yellowish white, nodular mass which completely occluded the bile duct. The common hepatic duct above this point was markedly dilated. The common bile duct, distal to this point was empty, somewhat shrunken in size, but patent. The left and right hepatic bile ducts were normal except for considerable dilatation. There was an enlarged, blue-gray, partially cystic mass behind the common duct in the region of the head of the pancreas, which appeared to be either a cyst or an enlarged group of lymph nodes. The stomach and duodenum were normal, as was the pancreas. The liver was slightly enlarged and a little more brownish in color than usual. A diagnosis of carcinoma of the common bile duct and chronic calculus cholecystitis was made. Cholecystectomy and excision of the involved portion of the cystic, common bile duct and lower part of the common hepatic duct were performed en bloc. An end to side choledochoduodenostomy was then performed between the common hepatic duct and the duodenum, completing the anastomosis over a small rubber tube.

The pathologist's microscopic report was as follows: "It [the tissue submitted] consists of masses of compact, highly cellular, anaplastic cells, apparently of epithelial origin. They are arranged in alveolar formation and the individual cells have large vesicular nuclei which are deeply staining. The exact origin of these cells is not clear, in view of the fact that they are highly undifferentiated. Pathologic diagnosis: Single gallstone, chronic cholecystitis, lymph nodes showing hyperplasia and edema. Anaplastic epithelial tumor compatible with a tumor from the common duct, anaplastic carcinoma." These slides were reviewed prior to publication of this paper by both the hospital and other pathologists and they concur in the diagnosis of carcinoma of the common bile duct.

The patient made an uneventful recovery with the exception of a temporary biliary fistula in the immediate postoperative period. She was last examined on August 13, 1957. At that time, she was well and free of any demonstrable recurrence of her carcinoma.

COMMENTS

The incidence of carcinoma of the extrahepatic bile ducts has been variously reported from 0.0135 to 0.895 per cent of autopsies. Goldenberg [3] found thirty-three instances of this disease in 8,131 autopsies, or 0.4 per cent. The incidence in 1,789 operations upon the biliary tract was 1.8 per cent. Kirshbaum and Kozoll [4], reviewing 13,330 consecutive autopsies at the Cook County Hospital, Chicago, Illinois, from January 1929 to April 1940 noted sixty-two patients with carcinoma of the extrahepatic bile passages. Neibling, Dockerty and Waugh [7] reported that there were sixty-six patients with this disease in 14,000 operations upon the biliary tract at the Mayo Clinic, or one in each 210 operations. The same authors reported an incidence of 0.26 per cent in 12,000 autopsies. Stewart [10] noted three instances of this disease in 22,512 autopsies at the Philadelphia General Hospital from January 1, 1920, to December 31, 1936, an incidence of 0.0135 per cent. Sako, Seitzinger and Garside [9] reported an autopsy incidence of 0.012 to 0.458 per cent. They stated that the disease occurred in 0.007 to 0.041 per cent. Kumayti et al. [5] found sixty-three instances in 24,029 autopsies at the Mayo Clinic from 1915 to 1954. This represents an autopsy incidence of 0.26 per cent. Glenn and Hays [1] reported that 6 per cent of all deaths in this country are due to cancer of the extrahepatic bile ducts. They gave the ratio of carcinoma of the gall bladder to that of the bile ducts as 6:5.

Men were more often affected than women, with a ratio of 2:1. The average age has been reported as fifty-five to sixty years. Stewart [10], however, found instances of the disease in young people, twenty-nine, thirty and thirty-two years of age. The most common location in the bile passages has been in the region of the common hepatic duct and the confluence of the hepatic, cystic and common bile ducts. The exact origin in the ducts is often difficult to determine. Gallstones have been frequently found associated with this disease, but not as commonly as in carcinoma of the gall bladder. Their presence has been reported in between 20 and 41.3 per cent of patients with carcinoma of the bile passages.

The pathology of carcinoma of the bile ducts has been described by several authors. Martin and Page [6] described three varieties: (1) villous or papillary, (2) nodular, and (3) diffuse fibrotic. The lesion may be a well localized small nodule or papilloma. Many patients presented a diffuse, dense, fibrotic type of carcinoma which tended to extend along the ducts in various directions. Both types may easily obstruct the lumina of the ducts. The neoplasm is most frequently an adenocarcinoma. Squamous carcinoma has also been reported by Sako, Seitzinger and Garside [9]. The disease spreads along the ducts to the liver and to the regional lymph nodes. Distant metastases were uncommon, although the authors just cited found metastasis in a cervical lymph node in one patient.

The most common symptom of this disease is jaundice. Abdominal pain was present early in the disease, but was not severe. Jaundice may be intermittent, and Kumayti et al. [5] stated that it fluctuated in 58 per cent of their patients. Eventually, it steadily progresses. There was moderate loss of weight in about half of the patients. Loss of appetite commonly occurred. The feces were often found to contain occult blood. The liver and gall bladder were, at times, enlarged and palpable. Altered laboratory examinations were essentially those of the obstructive jaundice and mild anemia. Accurate preoperative diagnosis was difficult and has seldom been made.

The treatment of cancer of the extrahepatic bile ducts has been symptomatic, palliative or radical. The results of treatment have been uniformly poor. Glenn and Hill [2], as well as others, believed that radical surgical procedures were least applicable in patients with this disease. Most operations must be looked upon as palliative, rather than curative. Local block resection with end to end anastomosis or rerouting between the bile passages and intestinal tract was often possible. Such resections have occasionally produced long periods of survival. Renshaw [8] has reported survival from such operations for fifteen months; and Neibling, Dockerty and Waugh [7] for two years and two months and Stewart [10] for twenty-seven months. Glenn and Hill [2] re-

ported on a patient who lived for three years. Goldenberg [3] had a patient who survived six years following palliative surgery. Such results are very infrequent and eventually the disease recurs and proves fatal.

Carcinoma of the common bile duct appears, therefore, to be an uncommon entity, justifying diagnostic consideration in the jaundiced patient and, at times, amenable to palliative treatment.

In conclusion, a case of carcinoma of the common bile duct is reported. The patient was treated by palliative local resection of the growth and has remained well for three years. The occasional long survival following palliative treatment of this disease is exemplified by this patient.

REFERENCES

1. Glenn, F. and Hays, D. M. Carcinoma of the extrahepatic biliary tract. *S. Clin. North America*, 33: 479, 1953.
2. Glenn, F. and Hill, M. R., Jr. Extrahepatic biliary tract cancer. *Cancer*, 8: 1218, 1955.
3. Goldenberg, I. S. Carcinoma of the biliary tract. *Am. J. Surg.*, 86: 292, 1953.
4. Kirshbaum, J. D. and Kozoll, D. D. Carcinoma of the gall bladder and extrahepatic ducts. A clinical and pathological study of 117 cases in 13,330 necropsies. *Surg., Gynec. & Obst.*, 73: 740, 1941.
5. Kumayti, K., Bagganstrosse, A. H., Stauffer, M. H. and Priestley, J. J. Carcinoma of the major intrahepatic and the extrahepatic bile ducts exclusive of the papilla of Vater. *Surg., Gynec. & Obst.*, 104: 357, 1957.
6. Martin, W. F. and Page, G. D. Carcinoma of the extrahepatic bile duct. *South. M. J.*, 44: 109, 1951.
7. Neibling, H. A., Dockerty, M. B. and Waugh, J. M. Carcinoma of the extrahepatic bile duct. *Surg., Gynec. & Obst.*, 89: 429, 1949.
8. Renshaw, K. Malignant neoplasms of the extrahepatic biliary ducts. *Ann. Surg.*, 76: 205, 1922.
9. Sako, K., Seitzinger, G. L. and Garside, E. Carcinoma of the extrahepatic bile ducts. *Surgery*, 41: 416, 1957.
10. Stewart, H. L. Carcinoma of the extrahepatic bile ducts. *Arch. Surg.*, 41: 662, 1940.

An Unusual Late Sequela Following Gastrectomy

THOMAS C. CASE, M.D., *New York, New York*

From the Department of Surgery of St. Vincent's Hospital, New York City.

IN recent years, with the increase in the volume of gastric resections, there has been an increasing awareness of the possible sequelae following the procedure. The more common sequelae are the dumping syndrome, the macrocytic anemia which follows extensive gastric resections and the efferent and afferent loop syndromes, all of which have been extensively described in the current literature. The loop syndromes are the result of improper technic, and while the complication that occurred in the case presentation to be reported is not really one of faulty technic, it should be considered one of incomplete technic.

The occurrence of the complication is exceedingly rare but can at times be fatal and therefore seems worthy of publication since closure of the gap or site of herniation is quite simple and should probably be performed as a routine procedure.

CASE REPORT

N. M., an adult white woman of sixty-one years of age, was admitted to St. Vincent's Hospital in New York City, March 17, 1957, because of repeated attacks of generalized abdominal pain and nausea, and this was occasionally accompanied by diarrhea. These episodes occurred soon after a gastric resection had been performed for a benign gastric ulcer in September 1953. At that time an antecolic type of Pólya left to right anastomosis was performed.

The episodes of generalized abdominal pain, which were crampy in character, started three months after the original operation to be more specific and, while the patient might be completely free of pain at some time she would never go more than several weeks without recurrence of the condition. On more than one occasion active peristalsis

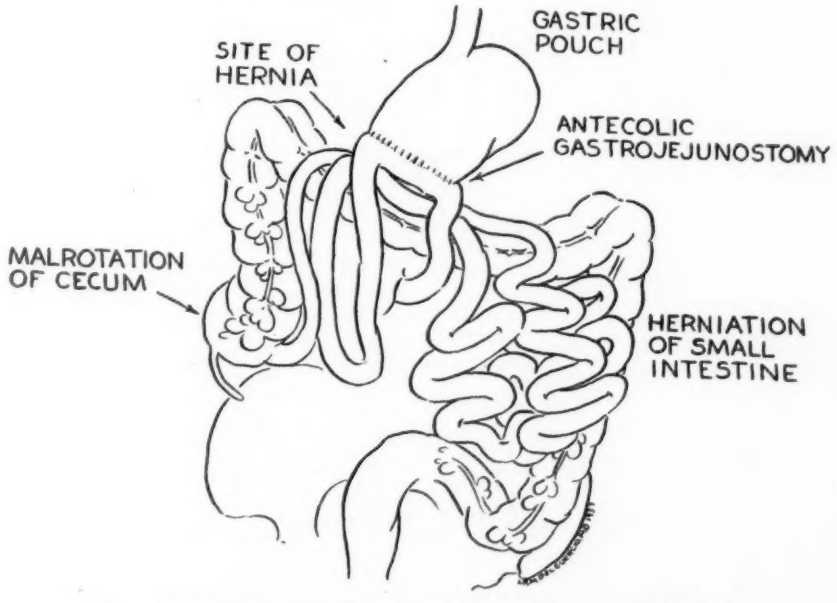


FIG. 1. Schematic representation of herniation of small bowel.

could be visualized throughout the surface of the abdominal wall.

The patient was constipated most of the time requiring cathartics in order to maintain normal bowel hygiene. However on many occasions the abdominal cramps would soon be followed by diarrhea.

X-ray examination of the upper and lower gastrointestinal tract revealed the presence of a redundant sigmoid and high cecum. At one of the examinations ileitis was suspected.

The patient had not put on any weight since operation. The hemogram revealed the presence of microcytic anemia. At no time was there any blood in her stool.

On March 19, 1957, it was decided to perform an exploratory celiotomy. At operation the entire small bowel was found to be herniated through the gap between the gastrojejunostomy anteriorly and the transverse colon posteriorly. (Fig. 1.) The cecum had not completely descended into its normal position and the mesentery of the small bowel was quite long. The bowel was not incarcerated because of the large opening present.

The opening was closed off with interrupted silk sutures after the small bowel had been replaced in its normal position. The sutures were placed between the appendices epiploicae, omentum and efferent portion of the gastrojejunostomy.

The patient made an uneventful recovery, has gained ten pounds in weight since the operation and has no further abdominal complaints.

COMMENT

Massive herniation of the small bowel through the ring formed by a gastrojejunostomy is a relatively rare sequela. It can be an extremely dangerous complication and has been reported as a highly [1] fatal type of internal hernia. The incidence of this condition may be expected to increase since the performance of gastric resections has increased tremendously in the past twenty years.

The herniation may occur from the day following [3] operation or perhaps many years after the original procedure had been performed as illustrated in this case report, and the amount of bowel that can herniate varies from a small segment to the entire small intestine.

Progress in surgery is in part a matter of foreseeing complications, since that is the surest way of avoiding them. Closure of the opening for the potential hernia is simple and should perhaps be performed as a routine procedure [2] in order to avoid this possibly fatal type of sequela.

SUMMARY

1. A case report of an unusual type of internal hernia which followed a gastric resection is presented.

2. Closure of the opening should be considered as a routine procedure in order to avoid this sequela.

REFERENCES

1. Wolfson, A. A highly fatal type of internal hernia. *Am. J. Surg.*, 91: 298–300, 1956.
2. Stammers, F. A. R. Small bowel obstruction following ante-colic, partial gastrectomy. *Brit. J. Surg.*, 40: 58–60, 1952.
3. Cannon, J. A. and Weeks, W. H. Complications of internal hernia ring routinely left unclosed in gastroenterostomy. *Ann. Surg.*, 138: 772–778, 1953.

BOOK REVIEWS

A Practical Handbook of Midwifery and Gynaecology. By W. F. T. Haultain, O.B.E., M.C., B.A., M.B., B.CH., F.R.C.P. (Ed.), F.R.C.S. (Ed.), F.R.C.O.G. and C. Kennedy, M.B., F.R.C.S. (Ed.), CH.B., F.R.C.O.G. Fifth edition, 410 pages, 48 illustrations. E. & S. Livingstone Ltd. Edinburgh and London, 1957. *Price* $7.00.

Students, interns and general practitioners should find this a good handbook for quick reference and a concise review of the basic principles required in the practice of normal and abnormal obstetrics and in the approach to gynecological problems. The volume is in semi-outline form and its size prevents extensive coverage of each subject.

Readers in America may question the fact that in so recent a publication little is said about the role of some of the newer hypotensive drugs in the treatment of pre-eclampsia and eclampsia; that little is said about the use of the broad-spectrum antibiotics in the treatment of infection. In addition, no mention is made in their section on the pregnant cardiac patient that the twenty-eighth to thirty-second week period is equally or more serious relative to the danger of decompensation than the period after delivery.

There is a good section on the management of the infant and neonatal conditions that should be welcomed by the general practitioner.

N. P. LIVINGSTON, M.D.

Chemistry and Biology of Mucopolysaccharides, Ciba Foundation Symposium. By G. E. W. Wolstenholme, O.B.E., M.A, M.B., B.CH. and Maeve O'Connor, B.A., Editors. Little, Brown and Company. Boston, 1958. *Price* $8.50.

The mucopolysaccharides represent a group of biologically important but chemically ill defined macromolecules consisting of a variety of carbohydrate and amino acid moieties. The wide biological distribution of mucopolysaccharides bespeaks of their physiological significance.

The present edition is the report of a Ciba Foundation Symposium held April 23–25, 1957. Individual reports of research and study in the field of mucopolysaccharides, encompassing their general chemistry; the specific chemistry of individual members; their biological distribution; their biosynthesis; their physiological, immunochemical and pharmacological effects, are presented by outstanding authorities. Although research in mucopolysaccharides is not of recent origin, the obvious incompleteness of the excellent work reported indicates that final understanding of this important group of compounds must await future effort. The book represents a helpful contribution to a dynamic field of current investigation.

R. O. MOORE, M.D

Transactions of the International Society of Plastic Surgeons. Williams and Wilkins Company. Baltimore.

This volume represents the papers presented at the First Congress of The International Society of Plastic Surgeons at Stockholm, Sweden, in 1955. Thirty-nine countries were represented at the Congress and papers were presented by plastic surgeons from many of these countries.

The papers are grouped into fourteen chapters. Particularly detailed are the chapters on burns and repair of the cleft lip and palate. Reconstructive procedures of the jaws, nose, eye, ear, scalp and cranium, esophagus, breast, abdomen, genitals and hand are presented. A chapter containing a miscellaneous group of interesting plastic procedures is included. A particularly intriguing chapter is devoted to clinical and experimental research.

The final chapter summarizes the surgical principles and technics involved in a variety of operative procedures, presented by means of motion pictures at the Congress.

Although the volume is written in English, there is a translation of the summary of each paper into French, German and Spanish.

BRUCE C. MARTIN, M.D.

Symposium on Diseases and Surgery of the Lens. By G. M. Haik, M.D., F.A.C.S. (Editor) and E. M. McFetridge, M.A. (Associate Editor). (D. Alvarado, Art Editor.) C. V. Mosby Company. St. Louis, 1957. *Price* $10.50.

This 260 page text presents the material delivered at the 1956 Symposium of the New Orleans Academy of Ophthalmology. Contributions include Cordes, Thorpe, Vail, Chandler, Dunnington and Irvine. The development of the normal lens and its cataractous derangements are thoroughly presented. Indications and technics are given for surgery in congenital, complicated and uncomplicated cataracts. The course of normal healing is described and followed by consideration of surgical and post-surgical complications. Vitreous changes, uveitis and glaucoma are discussed in relation to cataract. The book closes with a series of interesting round table discussions. It is well illustrated and presents a great deal of material in a readable style.

WILLIAM H. HAVENER, M.D.

Cortisone Therapy. By J. H. Glyn, M.A., M.D., M.R.C.P., D.Phys. Med. Philosophical Library, Inc. New York, 1957. *Price* $10.00.

This book is a relatively brief manual of well under 200 pages. It is written briefly, almost in outline form and yet, surprisingly, is skillful enough to be quite readable.

The author is predominantly interested in arthrology and rheumatology, and the book is for the most part written from this limited point of view.

The sections on history, chemistry, general pharmacology and physiology are well prepared outlines influenced by the author's interest in rheumatology.

The sections on rheumatology are apparently authoritative but basically of little interest to the surgeon. The sections on non-rheumatic diseases are scanty and admittedly not authoritative. They are basically student outline series level.

The book has an appendix of technics for intra-articular injections which is in outline form and may be of some value to the surgeon.

ROBERT N. WATMAN, M.D.

Roentgen Diagnosis of Abdominal Tumors in Childhood. By Charles M. Nice, Jr., M.D., PH.D., Alexander R. Margulis, M.D. and Leo G. Rigler, M.D. Charles C Thomas, Publisher. Springfield, Ill., 1957. *Price* $4.00.

This concise, well documented and profusely illustrated booklet deals with the diagnostic enigma of an abdominal mass in an infant or child.

The introduction wisely emphasizes the urgency and need for accuracy in preoperative diagnosis. After summarizing the developmental, anatomical and pathological facets of this problem, a method of establishing a diagnosis based upon simple conventional roentgen examinations is outlined. Complicated procedures are in general unnecessary and to be avoided.

The remaining chapters are devoted to discussion and radiographic illustrations depicting specific mass lesions which will be encountered in such studies.

This small volume is not only a practical clinician's reference but also an authoritative source for teaching material